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ANNALS
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I.

THE TECHNIC OF EXAMINATION OF THE STATIC LABYRINTH.

BY

ISAAC H. JONES, M. D., AND LEWIS FISHER, M. D.,

PHILADELPHIA.

So little has been known of the physiology of the static labyrinth until comparatively recent times, that the lack of a well established technic of its examination is not surprising. The value of the contributions of Ewald, Högjes, von Stein, Alexander, Neumann, Shambaugh, E. R. Lewis, J. G. Wilson, Ruttin, and others is now, however, beginning to receive the recognition it deserves. By far the greatest impetus to this work was given by Robert Bárány, who in the last decade brought out the "caloric" and "pointing" tests—contributions which brought him the Nobel prize. With these tests came the realization of the intimate relation of the internal ear to the rest of the central nervous system. We know now that the static labyrinth is only the labyrinth of the vestibular apparatus—this vestibular apparatus consisting of the static portion of the internal ear and nerve paths connecting it with nerve centers in the brain stem, cerebellum, and cerebrum.

Originally, tests of the labyrinth were carried out for the sole purpose of determining its own integrity. Such tests were sufficient for the aural surgeon who was called upon to decide whether or not to operate on the "end organ." With the development of the idea that the labyrinth is only one portion of the vestibular apparatus, came the realization that this whole apparatus was being tested at the same time that the internal ear itself was being tested, and that an intelligent interpretation of the phenomena obtained by such tests can also give the examiner an insight into the condition of those various brain paths and brain centers in relation with the internal ear. Thus, when a known stimulus is applied to the labyrinth, any response obtained therefrom, be it nystagmus or vertigo, indicates not only a functionating and reacting labyrinth, but also intact pathways from the labyrinth to the brain centers responsible for those reactions. Conversely, the nonappearance of any of the normal responses to stimulation indicates an interruption at some point along the particular pathway that fails to produce that particular response. This made it evident that the old technic of examination of the static labyrinth was very inadequate, and Bárány elaborated a new technic. Recent investigations by writers in the department of neurootology at the University of Pennsylvania, under the service of Dr. B. Alexander Randall, have shown that the fibers from the socalled horizontal semicircular canal have an entirely separate course in the brain stem from those of the vertical semicircular canals, and while they all go to the cerebellum, we consider that those from the horizontal canal reach it by way of the inferior cerebellar peduncle, while those from the vertical canals go by way of the middle cerebellar peduncle. Bárány's technic, elaborated before such a differentiation was demonstrated, deals with the labyrinth as a whole, whereas in the light of this new knowledge, it at once becomes evident how important it is to examine each set of canals separately.

We also became convinced that nystagmus and vertigo are distinct and separate phenomena, and that, furthermore, the "past-pointing" of Bárány is not a "cerebellar pull," but is a cerebral phenomenon exclusively, and depends entirely on the vertigo induced by the ear stimulation. This again necessitated the modification of some of the existing tests as well as the employment of some new ones. Above all, it is of prime importance that the technic be accurate, complete, and painstaking, if the data obtained from such an examination are to be relied on. We would suggest that those who appear to be doubtful of the value of these tests must ascribe their disappointment to laxity in methods of conducting them. This point has been impressed on us by the statement of one of the leading men in the profession, who claims that the "Bárány tests" had been routinely employed in his cases and proved of no value. We found that he had used no turning chair. Another authority reported a case in which there was an absence of the normal past-pointing, and yet operation revealed a normal cerebellum. We found that this physician did have a turning chair, but that it was not equipped with a "stop pedal"; we have frequently seen cases examined without a stop pedal in which no past-pointing was obtained, and on testing them with our chair with the stop pedal, we were able to produce past-pointing of a foot or more.

The technic about to be described has been employed in the examination of hundreds of cases, and the deductions drawn from them have been verified in many instances by operations and autopsies.

When we first began examinations of the static labyrinth, a great difficulty was encountered in the recording of the findings, or rather in the lack of recording them. All who have done this work realize what an unspeakable nuisance it is to keep haphazard records, on slips of paper, perhaps, of the results obtained from the various vestibular tests. For example: "On turning to the right the patient's nystagmus was horizontal to the left of so many seconds, but his past-pointing was so much for the right arm to the right, and so much for the left arm to the right," etc., etc. We all know how hopeless it is to attempt the analysis of a case from such records. To obviate this difficulty we gradually formulated the accompanying chart, in which all the tests are outlined in the order in which they are usually undertaken, and which is so arranged that when properly filled in it shows all the vestibular data simply by a glance at one page. We think this chart is of so much help in the examination and diagnosis of a case that it is actually a part of the technic, and we will, therefore, describe it. (Figure 1.)

One side of the chart is devoted to miscellaneous details or such routine matters as are found on any chart, with particular emphasis on the examination of the cochlea. The other side is devoted exclusively to the vestibular tests, of which there are three divisions, as follows:

1. Spontaneous.
2. Turning.
3. Caloric.

There is a complete column for the "nystagmus," and another complete column for "pointing." This enables us to study the nystagmus as such by running the eye down the column to the left, showing first the spontaneous nystagmus, then the nystagmus after turning, and then after the caloric test. In the same way the pointing tests can be studied as such by following right down the page, first the spontaneous, then those after turning, and then after douching.

As a further aid there is a space reserved on the chart for summarizing the findings obtained after each form of examination. As we always look for the following phenomena—nystagmus, vertigo, past-pointing, and falling—they are indicated concisely, one underneath the other, in each one of the three subdivisions. The examiner, after he has turned the patient, simply summarizes by indicating "nystagmus normal" or "absent"; vertigo, either "normal" or "subnormal," or "exaggerated" or "absent," as the case may be, and so on in the same way under the other headings. When the chart is filled in as we have indicated, and an analysis of the case attempted for the purposes of diagnosis, all the examiner need do is to look at these different summaries under the three main subdivisions to get a bird's eye view, as it were, of the whole case.

We will imagine we are examining a patient and conducting the tests step by step, with the aid of the chart. The "miscellaneous side" of the chart is filled in first, taking up every heading indicated and filling it in with appropriate information—the name, the age, and so on, with emphasis on the history of dizziness, for the reason that there can be no impairment of the vestibular apparatus without dizziness as a symptom. The duration of the dizziness, its character, when it was first noticed, whether it came on gradually or suddenly, whether the attacks came on with sudden change of position—as, for example, on getting out of bed in the morning, or when washing the face—whether nausea or vomiting accompanied it—all are noted.

Staggering.—Did it come on suddenly or gradually? Is it constant or does it come on intermittently? Is its presence coincident with dizziness? Is it at any time severe enough to make the patient fall? If staggering and falling are present, is it always in the same direction?

Tinnitus.—The presence or absence of tinnitus is of considerable importance, since it might be an aid in the differential diagnosis between peripheral and central lesions. We would expect tinnitus to be present in affections of the labyrinth itself or in lesions of the socalled "end organ" type—whereas it would be more likely to be absent in those disturbances of the vestibular apparatus located within the brain.

Deafness.—Its duration, and whether the loss of hearing was gradual or sudden, are noted.

Nose and Throat.—A routine examination is made with a view of discovering some evidence of focal infection which might account for the presence of an irritative disturbance of the vestibular apparatus, should the rest of the examination point that way; also, for any abnormality which might throw some light on any possible intracranial condition, such as palsies of the tongue, pharynx, or vocal cords, anesthesias, or loss of the sense of taste or smell.

THE EAR.

We note the following:

1. Configuration of the auditory canals.

2. The presence or absence of a mechanical obstruction in the canals, congenital or acquired; of the latter there may be impacted cerumen, or other débris or polypi.

3. The presence of inflammation or suppuration.

4. Condition of the tympanic membrane. The length of time necessary to douche before a reaction appears may depend in a measure on the thickness of the drum head. Perforations are looked for, since a "dry perforation" would be a contraindication to douching.

5. Hearing function. This is of the utmost importance in all cases, because a knowledge of the condition of the cochlear labyrinth is frequently of the greatest help in the differentiation between an "intracranial' and an "end organ" lesion. The cochlea is philogenetically the younger and newer portion of the labyrinth, and, therefore, the weaker and less resistant to the action of toxins. A perfectly functionating cochlea would, therefore, per se, presuppose a normal static labyrinth.

The Fistula Test.—This is performed only in cases where there is a chronic suppuration of the ear, and can be carried out with the use of the Politzer bag.

We are now ready for the vestibular tests, all of them being outlined on the "vestibular" side of the chart.

Spontaneous Phenomena.

NYSTAGMUS.

The patient is instructed to look straight ahead of him at a distant point, as the effort at convergence when looking at a near object may obscure the findings to the extent of limiting or entirely effacing a nystagmus. The use of convex lenses serves three purposes: first, convergence is entirely impossible; second, the observer is able to view the eyes from in front; and third, the eyes are now much magnified. A spontaneous nystagmus of any form on looking straight ahead is always pathologic. The patient is then told to look to the extreme right and the extreme left. With an unintelligent patient this test is accomplished more easily by placing him in the revolving chair and have him fix his eyes on some distant point. The chair is then turned, first to the left and then to the right. In this way we revolve the body, as it were, around the eyes. A certain amount of lateral nystagmus, when looking to the extreme right or extreme left, is physiologic. It is pathologic only when it is of considerable amplitude or when there is a sharp difference betwen the nystagmus on looking to the right and on looking to the left. All forms of nystagmus become more pronounced when the individual attempts to look in the direction of the nystagmus. It is for this reason that the subject is made to look in various directions, as by so doing we are often able to bring out a pathologic nystagmus of an amplitude not large enough to become evident on looking straight ahead. Nystagmus is recorded by means of an arrow —a straight one for horizontal nystagmus, and a curved one for the rotary; it seems simpler, as suggested to us by George Mackenzie, to have the arrow point in the direction in which the examiner sees the nystagmus on the patient. With eyelids held wide apart, the patient is told to look directly upward and then downward, with a view of discovering a vertical nystagmus, if present. This is of the utmost importance for the reason that, as Bárány has pointed out, a spontaneous vertical nystagmus is invariably intracranial in origin; furthermore, in our experience a spontaneous vertical nystagmus has proven itself to be a pathognomonic symptom of involvement of the brain stem either by pressure or infiltration. If a vertical nystagmus is present it is recorded—the arrow pointing either up or down, as the case may be. Paresis or paralysis of any of the ocular muscles, as well as the ability to perform conjugate movements of both eyes, are noted.

SPONTANEOUS VERTIGO.

The patient is asked not merely whether he is "dizzy," but whether he has any sensation of turning. If the latter is present, careful inquiry is made whether or not the turning is systematized. By systematized vertigo is meant:

1. The patient feels he is going in a certain direction.
2. The outside world is moving in a certain direction around him, but he himself is remaining still.
3. He himself is moving in one direction, and the world about him is moving in the opposite direction.

SPONTANEOUS POINTING.

(Figure 2.)

The patient sits in the chair and is told to put his arm forward and point with his forefinger. The examiner holds out his own forefinger, and allows that of the patient to come in contact with his. The examiner steadies his arm against his own body and further steadies his outstretched forefinger by means of the other hand. The patient's forefinger is allowed to come in contact with the examiner's finger, after which the patient raises his arm, without bending the elbow, to the perpendicular position, and immediately brings it back to touch the examiner's finger. He is then directed to repeat this with his eyes closed. Should the patient find the finger, it is recorded under the column for pointing for the appropriate arm with a letter "T" (touched). Should he deviate either to the right or the left, the test is repeated a number of times to make sure that the failure is not the result of pure inattention, but that the deviation is constant and persistent in a certain direction. The distance of the deviation is recorded in inches, either to the right, or to the left, as the case may be, by noting under the appropriate pointing column—let us say "2 to R.," if the deviation happens to be two inches to the right, or "2 to L.," if the deviation happens to be to the left, etc. Should the patient fail to find the finger and "past-point" either to the right or to the left, his arm must never be pulled over towards the examiner's finger, if the test is to be repeated, because if this is done the patient finds out that he "past-pointed," and might make a conscious correction to overcome an actual tendency to "past-point." Instead, the examiner should again place his own finger under that of the patient. In taking the pointing, it is usually sufficient to test the pointing of the shoulder joint "from above," as described; the other forms of pointing, such as "shoulder from below," or "shoulder from the side," or pointing of the hips, are not performed routinely —these are undertaken only in cases where such extensive examinations appear to be necessary.

SPONTANEOUS FALLING.

The Romberg Test.—The patient is told to stand with heels and toes together and eyes closed. His station is noted. While in this position the head is quickly turned to the right, and observation made whether that had any effect upon the "station," or upon the direction of the patient's "falling," should he have any. The same observation is made after the head is quickly turned to the left. This test is of value in that patients with intracranial lesions always fall in the same direction, regardless of the position of the head, whereas a labyrinthine lesion causes falling in the direction of the affected ear.

"The Attempt to Overthrow."—This is Bárány's own "pelvic girdle test." The individual stands as before, with eyes closed, heels and toes together. The examiner grasps the patient at both shoulders, and attempts to overthrow him either to the right or the left, forward or backward, instructing the patient all the while to resist this attempt at overthrowing by a balancing movement at the hips. When the shoulders of the patient are moved towards the right, the pelvis should sway toward the left in an attempt to maintain equilibrium. On pushing the patient backward, the pelvis should move forwards, and so on. In this way the degree of freedom of pelvic movement is observed. A normal individual balances perfectly, and can lean in any of the four directions considerably before falling, whereas in one with an affection of the vermis of the cerebellum, the pelvis fails to compensate, and the patient falls over like a broomstick at the slightest touch.

Goniometer.—This consists of a movable platform so arranged that it can be tilted out of the horizontal plane. One end of the platform has a cord attached to it by means of which that end can be pulled down—the other end rising against a graduated perpendicular column. The patient stands in the center of this platform, either facing the examiner or at right angles to him. He is first tested with eyes open. One end of the platform is gradually lowered by pulling on the cord until the patient begins to sway, showing that he is about to lose his equilibrium. The other (perpendicular and graduated) end shows the number of degrees of tilting that were necessary before the patient began to lose his equilibrium. He is then tested in the same way with eyes closed. By means of the goniometer it is sometimes possible to bring out a latent tendency to fall not demonstrable in any other way. This is only occasionally useful.

Having completed the examinations for the various spontaneous phenomena, we are now ready to examine the vestibular apparatus by means of the turning and caloric tests. In order to carry out a good technic, it is well to bear in mind the whys and wherefores of the various tests. Both the turning and caloric tests depend upon the setting in motion of the lymph within the labyrinth. This circulation of the lymph stimulates the hair cells in the ampullæ of the semicircular canals, starting impulses which are transmitted by nerve paths to the corresponding centers in the brain. The principle underlying these tests is that a known stimulus applied to a normal labyrinth will produce definite phenomena if the nerve paths from such a normal labyrinth to their centers are all intact.

Before drawing conclusions, therefore, we must be certain of our technic—we must make sure that the proper stimulus was applied, and must forever bear in mind the particular nerve path we are employing in a given test. The following points should be remembered:

1. The desirability of testing only one set of canals at a time.
2. In the turning chair we test only those canals which are in the horizontal plane, or rather those out of the absolutely vertical plane.
3. The caloric test affects only those canals which are in the vertical plane, or rather those which are out of the horizontal plane.
4. Each canal, when stimulated, produces a nystagmus and a vertigo in its own plane.
5. The eyes are always drawn in the direction of the endolymph movement (this is the slow component).
6. The vertigo is always in the direction opposite to the endolymph movement.

The individual is placed in a smoothly revolving chair which has an adjustable head piece, so that the head can be comfortably fixed and held in any desired position. The chair must also have a mechanism whereby it can be instantly stopped by means of a foot pedal in a given position and rigidly held there. Without such an arrangement past-pointing tests cannot be made accurately. Bárány constructed a special chair for this purpose, and we were fortunate enough to receive one of these chairs only a few days before the outbreak of the war in Europe. (Figure 3.) As it was hardly probable that any more of these chairs could come out of Vienna for some time, several physicians interested in the work asked us to design a chair along similar lines. After we had used the Bárány chair for several months, the following changes suggested themselves and were incorporated in the new chair (Figure 4):

1. The back of the chair and the head rest are so constructed that the patient's head is placed immediately over the axis of turning. This is obviously of great importance. In the original Bárány chair the head revolves away from the center of turning, and describes a circle with a diameter of over a foot.

2. It is impossible in the Bárány chair to hold the head in a forward position. We therefore constructed an extra head piece to permit rotation with the head inclined forward. It is obvious that it is absolutely essential for the head to be held steady when being rotated in this position, and this cannot be accomplished without a special head bracket.

3. Instead of having a special handle for turning, as in the Bárány chair, the rod at the back of the chair is made slightly longer and a handle placed at the top. The extra handle was very annoying and interfered with the pointing tests of the right arm by its presence on the side of the chair.

4. The base is made much heavier than in the Bárány chair, in order that the patient may be rotated rapidly, if necessary, and yet not have the chair wobble; this gives both the patient and the doctor a greater sense of security.

5. The Bárány chair is bound together by a great many bolts. In order to take the chair apart, in case it is desired to move it, all these bolts must be undone. In the new chair there are no bolts and the parts are all welded; the chair consists of only two pieces—the seat and the base. This makes it more portable, and is a great convenienec when a patient has to be examined at a place where no such chair can be had. In constructing this chair we also aimed to make it suitable for use as a regular office treatment and operating chair, so that it would not require any extra room in the office.

TURNING.

The routine method is to test first for nystagmus, then for vertigo, and then for past-pointing. The horizontal canals are tested in the turning chair. To accomplish this the head must be secured in the head rest, tilted thirty degrees forward —the reason being that with the head perfectly erect, the external or socalled horizontal semicircular canal slants thirty degrees backward. If this precaution is neglected, the vertical canals also enter into the reactions.

NYSTAGMUS AFTER TURNING.

During the turning the patient's eyes are closed. The chair is rotated to the right ten times, at the speed of two seconds to each turn, making twenty seconds for the ten turns, after which it is stopped. The patient is told to open his eyes and look off at a distance. The "after-turning" nystagmus is then noted, including its direction, character, and duration. The time of the nystagmus is best taken with a stop watch, the watch being clicked and time counted from the moment the chair is stopped until the very last nystagmus twitch disappears, when it is clicked again and the number of seconds read off. It is recorded with a horizontal arrow pointing in the proper direction after the words "To Right" on the chart. The amplitude is recorded in terms of either "large," "small," "fair," or "barely a twitch." The duration is recorded in seconds as read off from the watch. In a normal case this nystagmus should be twenty-six seconds.

In a similar manner the patient is turned to the left.

The employment of the stop pedal is not necessary in testing for nystagmus.

VERTIGO AFTER TURNING.

Vertigo after turning may be tested quantitatively. The plane of the induced vertigo is always in the plane of the canal stimulated and in a direction opposite to the endolymph movement. If it is the horizontal canals we wish to test, the patient's head must be fixed with the chin tilted thirty degrees forward. He is then turned, with eyes closed, to the right at a uniform speed, and is asked to keep on telling the examiner in what direction he is being turned. Thus he keeps on saying "to the right, to the right." After ten turns in ten seconds the chair is stopped and immediately he will say, "I am going to the left." The stop watch is started the same instant and kept running as long as the patient thinks he is going to the left. When he says, "I am standing still," the watch is stopped and the reading of the duration of the vertigo taken in seconds. In a normal case the duration of the vertigo is approximately twenty-four seconds.

The test is performed in a similar way by turning the patient to the left.

This quantitative test for vertigo is not necessary as a routine procedure. "Past-pointing," which we are about to discuss and which is routinely tested, makes this unnecessary. When the patient is turned to the right and stopped, he feels that he is turning to the left, and for this reason he "past-points" to the right. That is, when the patient is stopped after being turned in the chair, he has a subjective sensation of turning in a direction opposite to that in which he was actually turned. After touching the examiner's finger at this time, he is under the impression as he is raising his arm that he is turning away from the finger, and therefore tries to correct for that by moving the arm out to the point where he conceives the finger now to be. Since as a matter of fact he is not moving, the chair being held rigidly still, he points widely past the finger and continues doing so in lessening degree as long as the vertigo lasts. The very presence, therefore, of past-pointing is indicative of the presence of vertigo —the past-pointing being the objective evidence of his subjective impression of turning.

PAST-POINTING AFTER TURNING.

The patient's head is again fixed so that the horizontal canals only are tested, and the chair turned at double the speed of that for nystagmus—that is, the ten turns are made in ten seconds instead of twenty. The patient is carefully instructed as to what is expected of him, and is told to keep his eyes closed throughout the entire test. The examiner stands in front of the patient with the stop pedal near his right foot. As the chair is turned for the tenth time the pedal is released by the examiner's right foot and the speed of the chair gradually slackened, so that the stop is accomplished without any jarring. The patient's right hand is then quickly grasped, and after his forefinger touches the examiner's finger, the examiner says "up," upon which the patient raises the arm in question to the perpendicular and immediatley tries to come back to the examiner's finger. The "past-pointing" is called off in inches and recorded by the assistant where it says "shoulder from above," as, for instance, "15 to R." (fifteen inches to the right). The left arm is immediately tested in the same way, then the right arm again, then the left arm again, and so on until there is no longer any past-pointing. In a normal case the vertigo lasts sufficiently long to permit of three past-pointings of each arm, gradually lessening in extent until the patient is again able to find the finger.

The patient is then turned to the left and the past-pointing of both arms taken and recorded as above.

The patient's eyes must be kept closed throughout these tests, and if his cooperation is doubtful, it is best to blindfold him.

The turning tests affect only those semicircular canals which are in a horizontal plane while the patient is turned. Should it be desired to test the two sets of vertical canals by turning, the patient's head must be placed either one hundred and twenty degrees forward or sixty degrees backward. For turning with the head forward, a small bar with a comfortable head rest is placed between the two arms of the turning chair, as shown in Figure 4, and the patient turned ten times, first to the right. The resulting nystagmus and vertigo are of course in the frontal plane—a rotary nystagmus to the left and vertigo to the left.

In taking the past-pointing the patient keeps his head in the same position after stopping the chair, and the examiner is forced to kneel on the floor in making the test. The past-pointing is not so large as that obtained from the horizontal canals, but is with both arms to the right after turning to the right, and with both arms to the left after turning to the left.

When testing the vertical canals with the head back, the upright, or turning, handle is loosened at the ratchet and turned back. The head rest is brought all the way down and the head comfortably fixed in a position sixty degrees backward. The patient is turned to the right, and there is produced a rotary nystagmus to the right (just the opposite to that obtained from turning with the head forward). The past-pointing, however, is also to the right, just the same as that obtained after turning to the right with the head upright or with the head forward. It may be remarked in passing that this test does not bear out the statement and law laid down in all the writings on this subject, including Bárány's, that "the past-pointing is always in the direction opposite to that of the nystagmus." Nystagmus and vertigo (with its consequent past-pointing) are not in any way dependent on each other. They are different reactions produced by stimuli sent along entirely different paths. A normal individual always past-points to the right after having turned to the right, regardless of the position of the head. Nystagmus, on the other hand, is in a diametrically opposite direction, after turning with the head backward from what it is on turning with the head forward. Turning to the right with the head back produces a nystagmus to the right and a past-pointing to the right, so that the statement that "past-pointing in a direction opposite to the nystagmus" is not only misleading and confusing, but actually incorrect.

FALLING AFTER TURNING.

This results when the individual tested has a subjective sensation of turning in a vertical plane. He will fall to the right or left if he thinks he is turning in a frontal plane, and will fall forward or backward if he thinks he is turning in a sagittal plane. Therefore, turning a patient with the head tilted thirty degrees forward produces no falling, as the patient feels he is revolving in a plane parallel to the floor. Similarly, turning with the head forward or backward produces no falling so long as the head is maintained in this position. If, however, after turning with the head forward or backward, the head is brought to the upright position, the subjective sensation is now one of turning in a plane at right angles to the floor, and the patient falls either to the right or left. Again, if the head is inclined toward the shoulder, after the turning the subjective sensation is one of turning in a plane parallel to the floor; if the head be raised to the upright position, however, the subjective sensation is that of turning in a sagittal plane, and the patient falls forward or backward. These tests, however, are seldom necessary in routine examination.

CALORIC TEST.

The main advantage of the caloric test of Bárány is that it enables us to examine each internal ear separately, and also to analyze the function of its canals separately, whereas turning stimulates both labyrinths at the same time. It is essential to have an absolute standard in the matter of temperature of the water. As Bárány directs, we employ water at 68° F.

This temperature is sufficiently cool to secure a good reaction, and yet not cold enough to be uncomfortable to the patient. When hot water is to be used the temperature is 112° F. The latter should be but seldom used.

The vessel containing the water is placed about two feet above the level of the ear to be douched. The shape or size of the nozzle is immaterial, the essential thing being that a free and continuous stream of water shall flow against the drum membrane maintaining an even temperature of sixty-eight degrees. The nozzle shown in Figure 5 is very convenient, as it has a closing valve within easy reach of the fingers. This valve also enables the examiner to regulate the force and volume of the stream of water entering the canal. Figure 6 shows a rubber receptacle placed underneath the patient's ear to catch the return flow of the water. A tube at the bottom conducts the water into a basin below. When this is properly attached there is little chance of wetting the patient, and if necessary douching can be kept up for several minutes without any interruption.

Remembering the desirability of testing only one set of canals at a time, and recalling still further that this caloric test influences only those canals which are in a vertical plane, the patient's head is placed thirty degrees forward so as not to include the horizontal canal in the reaction. The chair is held firmly by the foot clamp, the stop watch is held ready, the nozzle is inserted into the canal, and the instant at which douching is started the stop watch is clicked. The patient's eyelid is elevated; the patient is asked to look down and the eyeball carefully watched for any rhythmic nystagmus to appear. The moment that a rotary rhythmic nystagmus appears the stop watch is clicked again and the number of seconds necessary to produce the reaction noted. We usually continue the douching for a few seconds longer in order to obtain the maximum reaction. In the normal, nystagmus appears after forty seconds, and after douching for five or ten seconds more shows a large amplitude. The direction of the nystagmus is recorded by a curved arrow on the chart where it says "douche right." The amplitude is noted beneath and then the length of time required to produce it.

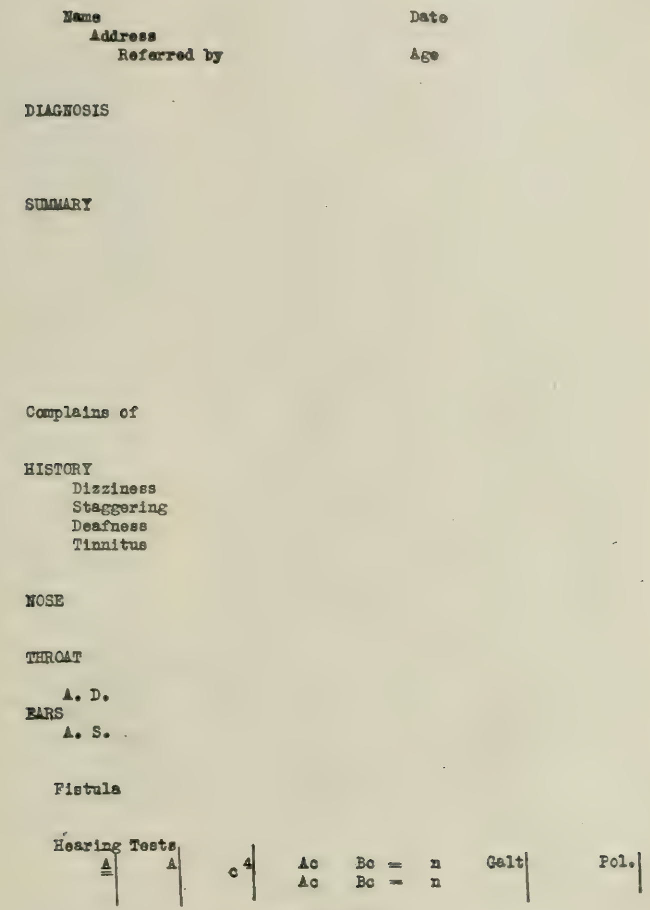
The patient is then told to close his eyes and the pointing tests are carried out and recorded in the same way as before described. It is well to remember that this douching has produced vertigo in the frontal plane—the same reaction that occurs after turning with the head forward; the patient, therefore, exhibits a marked tendency to fall, and it is advisable to have an assistant hold the patient's head firmly in the head rest while taking the pointing tests. As soon as the past-pointing of the arms has been taken, the head is quickly tilted to a position sixty degrees back, which places the horizontal canal in the vertical plane in a position in which it can be affected by the chilling. The patient is told to look upward, and the existing rotary nystagmus immediately becomes horizontal. The pointing of both arms is then quickly taken with the head in that position. The head may then be tilted forward ninety degrees and the past-pointing of both arms be taken again. This new position of the head again influences the horizontal canal, but has reversed the direction of the endolymph movement. All the reactions, therefore, are also reversed. The duration of the average stimulus after douching is long enough to permit of the examination of the head in the three positions just given. It is well to note that in this way one douching of only one ear can test out all the pathways for nystagmus as well as produce past-pointing in all directions, enabling us to test out the integrity of the entire cerebellum.

Falling.—The caloric test by its very nature produces vertigo only in a plane at right angles to the floor, so that "falling" always occurs on douching a normal ear. All that is routinely necessary is to observe the tendency to fall, while the other examinations are going on, and to note it on the chart.

ELECTRICAL.

For practical purposes the electrical tests have been of only slight use in our experience. Since the ability to examine one set of canals at a time is of such prime importance in making these tests of great clinical usefulness, it is evident how limited are the uses of an agency which stimulates at the same time not only the entire labyrinth, but the eighth nerve as well, and perhaps even the medullary nuclei. It is of great use, however, when the question arises as to a differential diagnosis between a destruction of the labyrinth and the eighth nerve. In a recent destruction of the labyrinth the caloric test will produce no reactions. The electric current, however, may directly affect the eighth nerve and produce normal reactions. When the galvanic current is used a large electrode is held in one hand, and a small one is placed on the mastoid process. Both electrodes should be covered with cloth or cotton fairly saturated with a normal salt solution. The current is gradually turned on, and when four milliamperes are discharged a nystagmus should appear.

It must not be thought that a routine examination of the internal ear requires all the preceding tests. In order to illustrate the simplicity of a complete average examination of the static labyrinth we insert a chart properly filled in with the results of the examination of a normal individual (Figure 7). Naturally, for example, if douching the vertical canals of both ears gives a doubtful response, it is wise to verify the examination by turning with the head back—or forward; in this case the findings are recorded on an extra chart to avoid confusion. In the average case, however, it is necessary merely to fill out one vestibular page, as shown, and to conduct all the tests with the head upright; the turning in this position tests the horizontal canals, and the douching examines the vertical canals. The examiner needs merely to complete this chart to have before him all the essential data of a routine examination.



Name Date
Address
Referred by Age

DIAGNOSIS

SUMMARY

Complains of

HISTORY
Dizziness
Staggering
Deafness
Tinnitus

NOSE

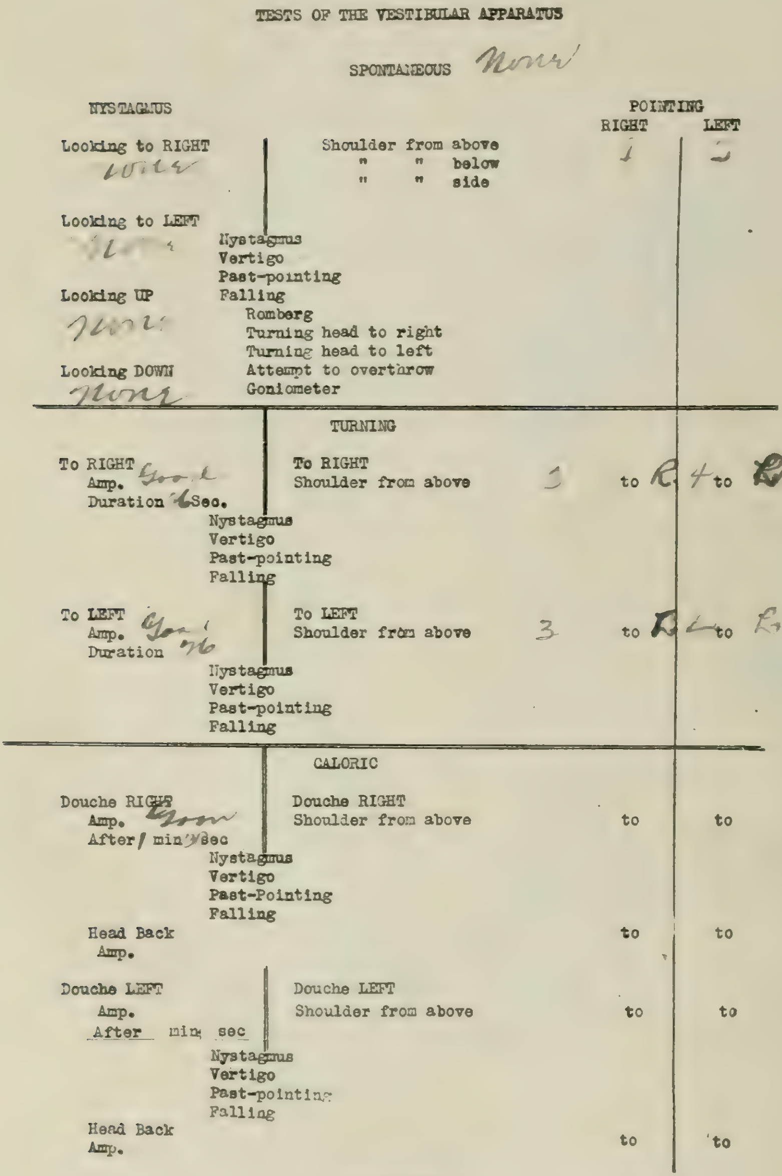
THROAT

A. D.
EARS
A. S.

Fistula

Hearing Tests
$\underline{\underline{A}}$ | A | c^4 | Ac Bc = n Galt | Pol. |
Ac Bc = n

FIGURE 1.



TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS None

NYSTAGMUS

POINTING
RIGHT LEFT

Looking to RIGHT
Wide

Shoulder from above 1 2
" " below
" " side

Looking to LEFT
None

Nystagmus
Vertigo
Past-pointing
Falling
Romberg
Turning head to right
Turning head to left
Attempt to overthrow
Goniometer

Looking UP
None

Looking DOWN
None

TURNING

To RIGHT
Amp. Good
Duration 26 Sec.

To RIGHT
Shoulder from above 3 to R. 4 to R.

Nystagmus
Vertigo
Past-pointing
Falling

To LEFT
Amp. Good
Duration 26

To LEFT
Shoulder from above 3 to R 2 to R

Nystagmus
Vertigo
Past-pointing
Falling

CALORIC

Douche RIGHT
Amp. Good
After 1 min 3 sec

Douche RIGHT
Shoulder from above to to

Nystagmus
Vertigo
Past-Pointing
Falling

Head Back to to
Amp.

Douche LEFT
Amp.
After min sec

Douche LEFT
Shoulder from above to to

Nystagmus
Vertigo
Past-pointing
Falling

Head Back to to
Amp.

FIGURE 1.

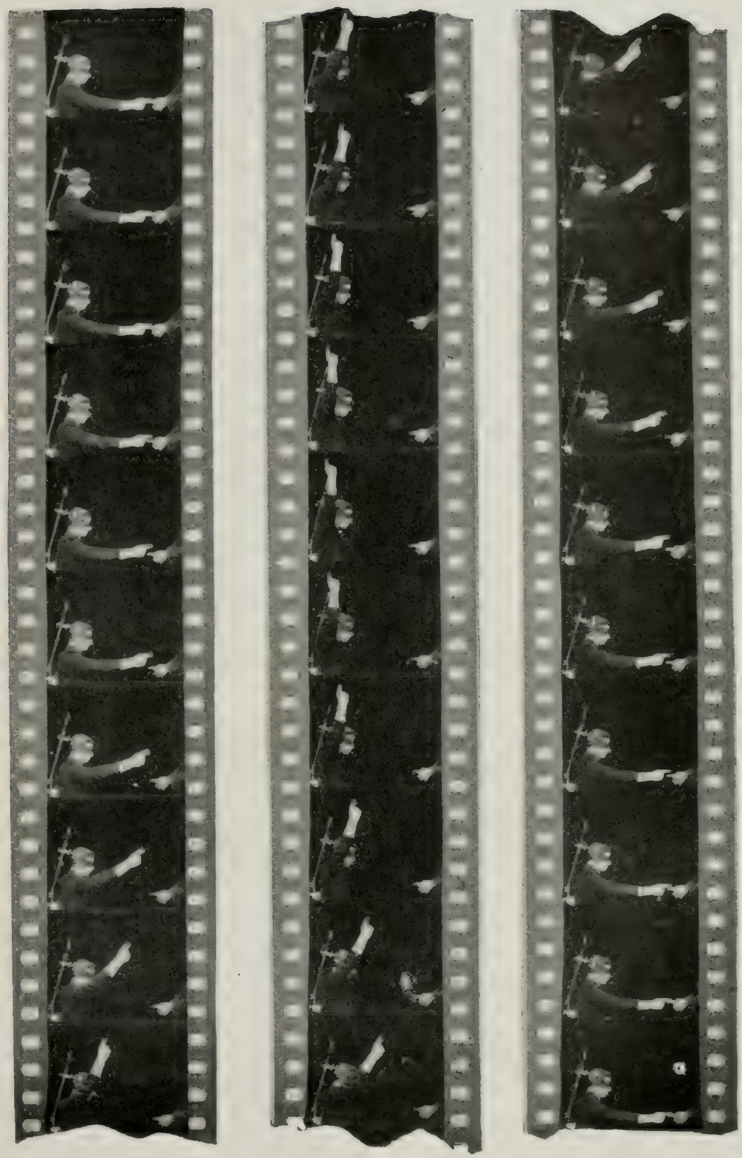


FIGURE 2.

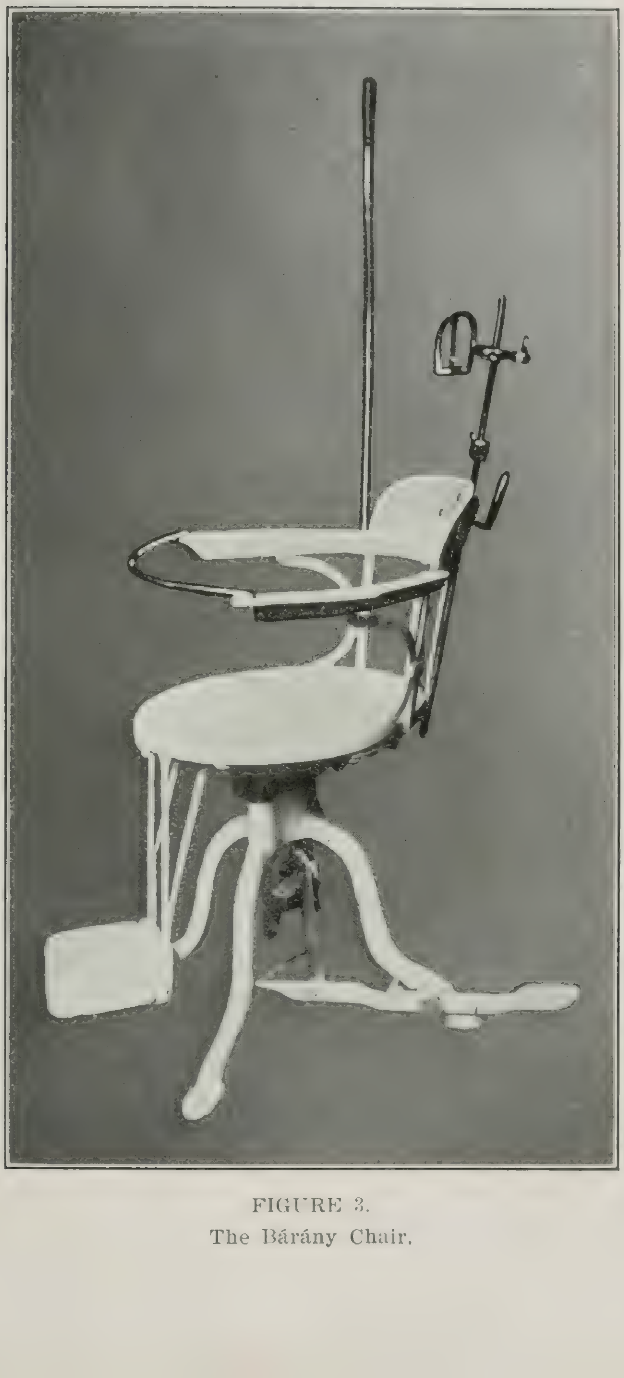


FIGURE 3.
The Bárány Chair.

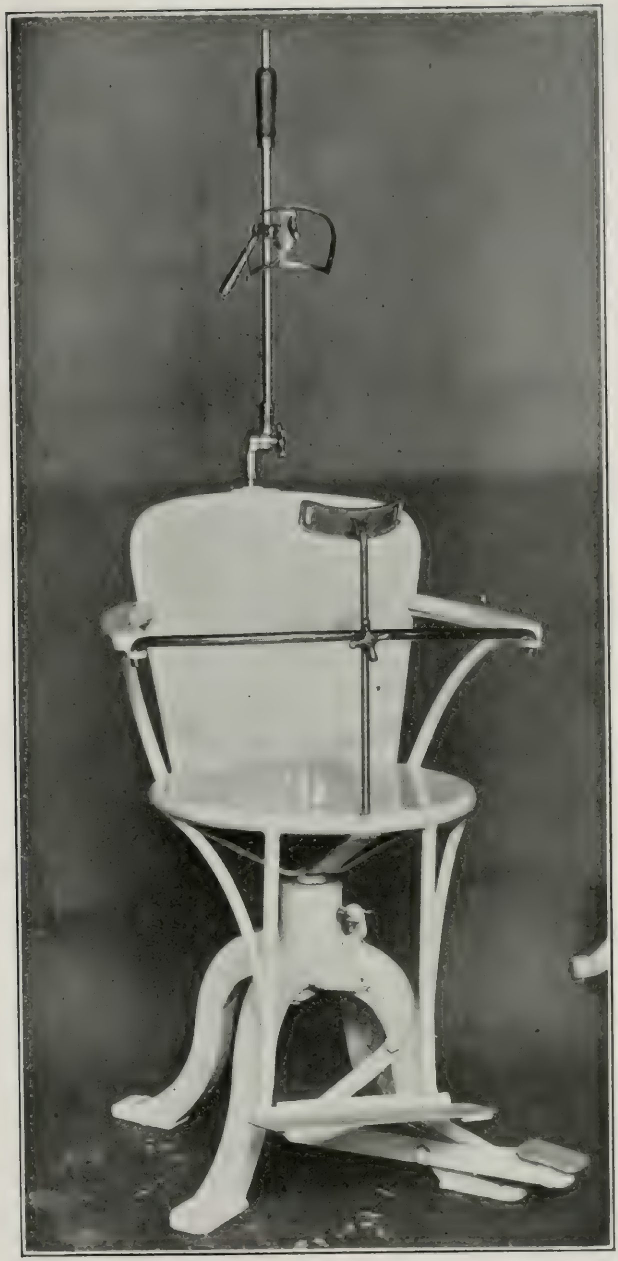


FIGURE 4.
American Modification of the Bárány Chair.

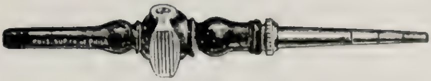


FIGURE 5.

Nozzle with closing valve.

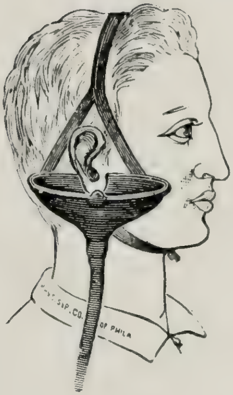
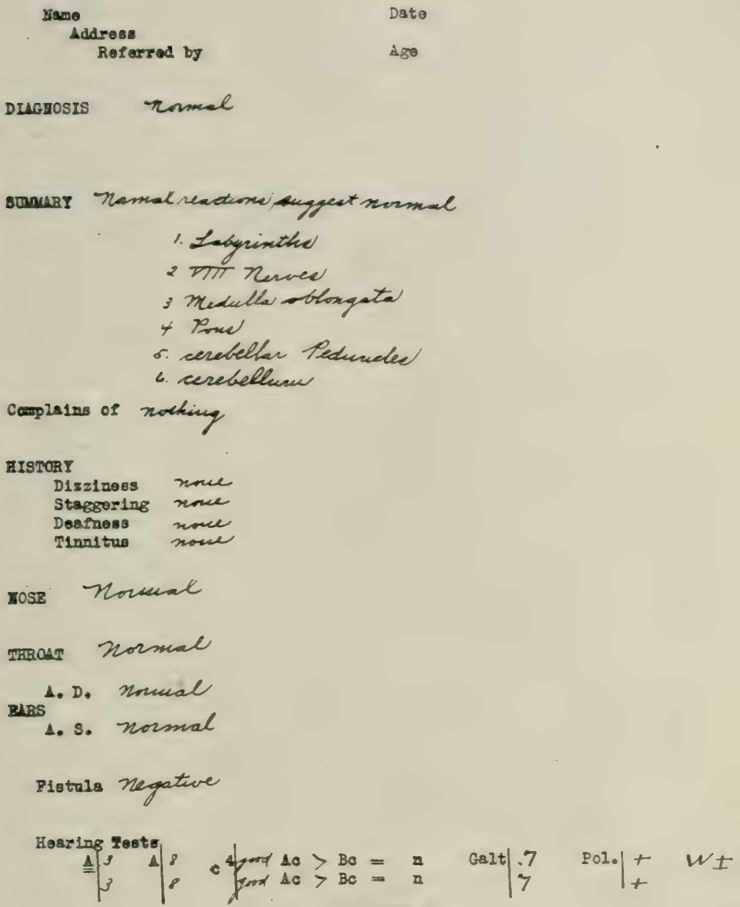


FIGURE 6.

Rubber receptacle to catch return flow of water.



Name
Address
Referred by

Date

Age

DIAGNOSIS Normal

SUMMARY Normal reactions suggest normal
1. Labyrinths
2 VIII Nerves
3 Medulla oblongata
4 Pons
5. cerebellar Peduncles
6. cerebellum

Complains of nothing

HISTORY
Dizziness none
Staggering none
Deafness none
Tinnitus none

NOSE Normal

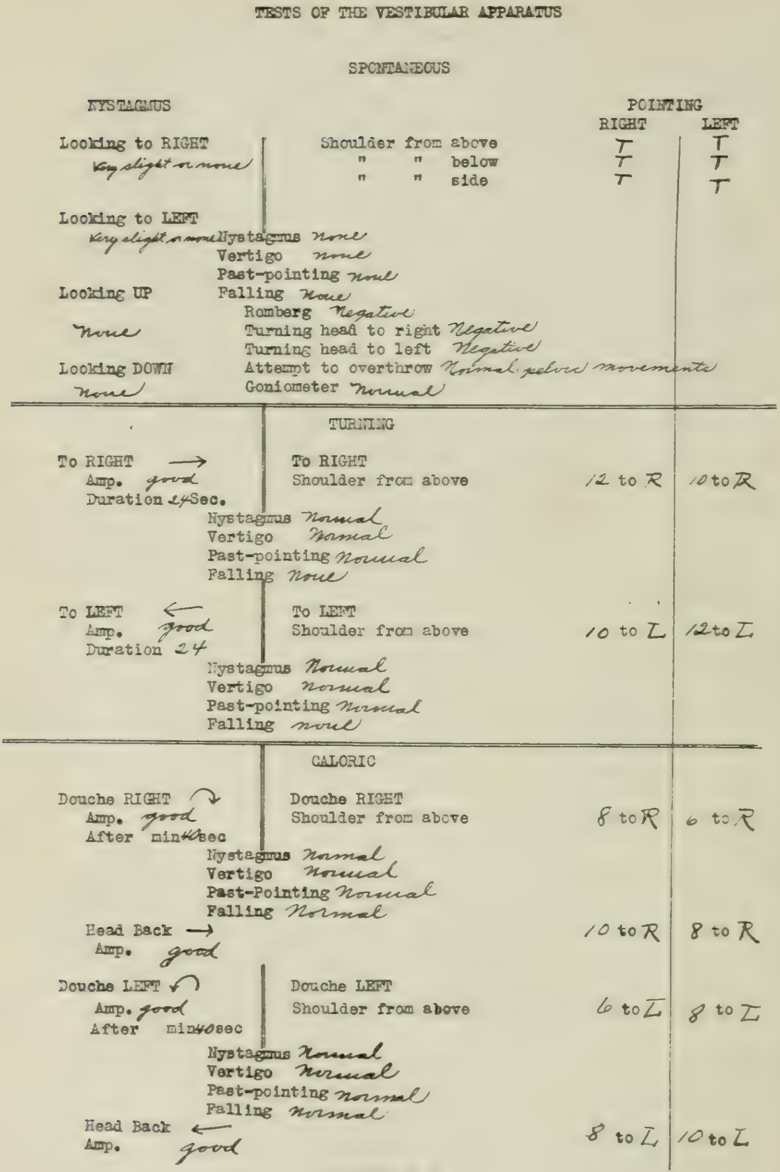
THROAT Normal

EARS
A. D. Normal
A. S. normal

Fistula Negative

Hearing Tests
A | 3 A | 8 c 4 good Ac > Bc = n Galt | .7 Pol. | + W ±
| 3 | 8 good Ac > Bc = n | 7 | +

FIGURE 7.



TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS

Looking to RIGHT
Very slight or none

	POINTING RIGHT	POINTING LEFT
Shoulder from above	T	T
" " below	T	T
" " side	T	T

Looking to LEFT
Very slight or none

Nystagmus None
Vertigo none
Past-pointing none
Falling None
Romberg Negative
Turning head to right Negative
Turning head to left Negative
Attempt to overthrow Normal, pelvic movements
Goniometer Normal

Looking UP
None

Looking DOWN
None

TURNING

To RIGHT →
Amp. good
Duration 24 Sec.

To RIGHT	RIGHT	LEFT
Shoulder from above	12 to R	10 to R

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling None

To LEFT ←
Amp. good
Duration 24

To LEFT	RIGHT	LEFT
Shoulder from above	10 to L	12 to L

Nystagmus Normal
Vertigo normal
Past-pointing Normal
Falling none

CALORIC

Douche RIGHT ↷
Amp. good
After min 40 sec

Douche RIGHT	RIGHT	LEFT
Shoulder from above	8 to R	6 to R
Head Back →	10 to R	8 to R

Nystagmus Normal
Vertigo Normal
Past-Pointing Normal
Falling Normal

Head Back →
Amp. good

Douche LEFT ↶
Amp. good
After min 40 sec

Douche LEFT	RIGHT	LEFT
Shoulder from above	6 to L	8 to L
Head Back ←	8 to L	10 to L

Nystagmus Normal
Vertigo Normal
Past-pointing normal
Falling normal

Head Back ←
Amp. good

FIGURE 7.

II.

AN ANALYTIC STUDY OF THE RINNE AND OTHER TUNING FORK TESTS.*

FIRST COMMUNICATION CONCERNING PERCEPTION DEAFNESS.

BY JESSE WRIGHT DOWNEY, JR., M. D.,

BALTIMORE.

I.

In a paper published in 1855, Rinné[1] claimed that a vibrating tuning fork is stronger and longer heard by bone conduction than by air conduction in diseases of the middle ear, and vice versa in diseases of the perception apparatus. The test did not come into general use until 1887, when Lucae[2] called attention to its great importance and elaborated many rules to govern its application, as likewise did Bezold.[3] An interesting résumé of the earlier controversies concerning the test was given by Turnbull and Reed[4] in 1888. From then to date contributions to the literature of the subject have been numerous.

It would be hardly possible to doubt the diagnostic value of the Rinné experiment in testing hearing, and yet there is no statement so misleading as the average textbook instructions concerning the method of comparing bone and air conduction. Nine out of ten authors describe the test as being made by holding the tuning fork over the mastoid until the patient says it is no longer heard, when it is then quickly transferred to the front of the auricle, and if the patient says that it is again heard, the test is positive, and the lesion, if one exists, is probably in the sound perceiving apparatus; if the tuning fork is not heard by air conduction, the Rinné is negative, and the deafness is conductive in character. The method of making the test may also be reversed.

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*Read before the Section on Ophthalmology and Otology of the Baltimore City Medical Society, October 18, 1916.

By the Rinné experiment we should attempt to determine:

(1) Whether or not air conduction is normal.

(2) Whether or not bone conduction is normal.

(3) In which course of sound conveyance lies the defect, or the greater defect, in hearing.

Under the first proposition we test all those structures which have to do with the conveyance of sonorous vibrations until they are perceived as sound. Under the second proposition we test those intralabyrinthine structures which have to do with the conversion of the mechanical vibrations into a nerve stimulus and the conveyance of this stimulus to the higher centers. The Rinné test should be made, therefore, as recommended by the Eighth International Otological Congress (Budapest, 1909), by a separate test of air and bone conduction, and the result recorded accordingly.

II.

In the first analysis we must consider sound with all its phenomena in terms of physical vibrations which would exist as a part of kinematics or mechanics, even if we did not have the sense of hearing. The ears convey and properly transpose, but they do not create, the vibrations which the brain recognizes as sound; therefore, to a large extent, hearing is governed by physical and anatomic properties rather than by physiologic or psychologic hypotheses. The vibrations set up in the air by any sonorous impulse—for example, a tuning fork—are taken up by the drum membrane as vibrations, and are conducted by the ossicular chain to be transmitted by the footplate of the stapes to the perilymph of the scala vestibuli; these vibrations continue around the helicotrema into the scala tympani to impinge, if there be sufficient force, against the membrane closing the foramen rotunda. During the course of the vibrations through the perilymph of the scala vestibuli they are transferred to the endolymph of the ductus cochlearis, probably by the law of physical resonance stimulating into vibration certain analytic areas in the membrana tectoria. As the result of the interaction between the membrana tectoria and the hair cells of the organ of Corti the vibrations are converted into a nerve stimulus, and this is transmitted by the cochlea branch of the eighth nerve to the cerebral centers in the temporosphenoidal lobes of the brain, and recognized, in the case of our example, as the sound of a tuning fork.*

From the foregoing it must be obvious that up to the point where the vibrations are converted into a nerve stimulus, hearing must be governed by the same laws governing physical vibrations. Elementary physics teaches that the pitch of a sound is due to the number of vibrations to the second set up by the initial shock, and that it is constant as long as the sound can be perceived; the intensity, or loudness, however, depends on the amplitude of the vibrations, and decreases from second to second until the oscillations are too small to be taken up and conveyed by the conduction apparatus, and then our perception of sound ceases. The conduction apparatus of the normal ear can convey sonorous shocks as slow as sixteen double vibrations to the second, and as rapid as thirty thousand or more, if the intensity is sufficient. All vibrations within this range are capable of setting up resonance in the tectorial membrane, to be taken up by the interacting hair cells of the organ of Corti and converted into a nerve stimulus and recognized as sound. Hence, hearing may be said to be measurable by the ability of the conductive apparatus to transmit and the nervous apparatus to perceive vibrations of any rapidity within the normal range under what is estimated to be a minimum intensity as compared to the normal minimum intensity. Therefore, it is not sufficient to determine that a tuning fork is "heard," for such a determination may only mask a degree of deafness which may be of diagnostic importance; likewise, the fact that a tuning fork is "not heard" may only mean that we are below that intensity necessary to set a defective conduction apparatus into vibration, or to stimulate a diseased nerve ending, or even to create a nerve impulse powerful enough to "get through" a true "nerve block."

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*Without entering into the mechanical action of the ossicles, this is the simplest physiologic explanation of the conveyance, the reception and the perception of sound vibration. It is the Helmholtz theory augmented by the investigations of Shambaugh,[5] who has anatomically demonstrated the impossibility of the basilar membrane being the vibrating structure, whereas the tectorial membrane apparently can and does fulfill this rôle. This theory is much more logical than the speculative "telephone theory" of Rutherford, and it will be dogmatically accepted throughout this paper.

III.

With a tuning fork we have a means of obtaining a musical tone of constant pitch with a prolonged intensity which decreases in definite retrogression until its vibrations can no longer be conveyed or perceived as sound. It is necessary that we make the attempt to determine the intensity under which a fork is heard, and, with all its faulty factors, our best index of this is the difference between the faulty duration of perception and the normal duration of perception; for it is the only practical way that we have of determining the vibrations of the smallest amplitude capable of setting up similar vibrations which can be conveyed and perceived as sound. Unfortunately, the decrement of the vibrations of a tuning fork is not uniform from second to second, but is much more rapid at the beginning than near the end, for the reason that the amplitude decreases by geometric progression; that is to say, the decrement in intensity from one step to the next takes place by a division of the initial and the succeeding amplitudes by a constant quantity. This constant quantity varies with each and every tuning fork, and otology is greatly indebted to Dundas Grant[6] for the methods and formulæ necessary to determine it. Though the problems involved almost prohibit the scientific estimation in everyday work of the actual percentage value of the hearing of the tuning forks, the law of decrement is unalterable, and the practical significance is as follows:

In all tests with all tuning forks the duration of the perception, both abnormal and normal, must be determined and noted, and the difference in time between defective and normal perception of the duration of the vibrations, and not the duration of perception itself, taken as the factor of importance and the one which must serve as the index of defective audition. (See Figures 1 and 2.)

The normal duration of perception will vary greatly, as it is practically impossible to obtain a standard initial intensity with the various forks, or even with the same fork at different times, though many methods have been tried, notably by Gradenigo,[7] whose investigations should be carefully studied, but the abnormal hearing will also vary in approximately the same proportion.*

IV.

Hearing by air conduction is physiologic, the mechanical perfection of the ossicular chain making it possible for us to interpret vibrations of very low intensity. Up to the point where the vibrations are transferred by resonance to the tectorial membrane, hearing is purely a question of sound wave conduction; beyond this point hearing becomes truly perceptive in character. In diagnosis it is important that we be able to locate the situation of a lesion causing deafness, and this becomes possible because a vibrating tuning fork placed upon any portion of the cranium can be perceived without the waves being conducted through the ossicular portion of the conduction apparatus.†

The normal duration of perception for a tuning fork by bone conduction is much less than the normal duration of perception by air conduction.‡ It seems to be generally assumed that this is so for the reason that sound vibrations are not transmitted as well through a solid as through the air. Without qualification, this is a makeshift and unscientific explanation, for sound vibrations are imparted to and conducted for some distance through solids without appreciable loss in the intensity. How then may an inch of porous bone, forming almost a tubular conductor, reduce the normal hearing by bone conduction to a minimum percentage value of the same sound conducted through the medium of the drum head and the ossicles?

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*In The Laryngoscope for September, 1914, I published the description of "An Apparatus Designed to Give an Approximately Accurate Quantitative Hearing Test." The instrument was constructed with the purpose of obtaining a tuning fork stimulation of uniform value, and hence a sound of constant intensity. The apparatus is more or less of a failure in this respect, but after hundreds of trials I have found that the proportion between abnormal and normal hearing is strikingly preserved, though the duration of perception may vary considerable.

†Bezold and Henson claimed that the oscillations were first taken up by the tympanic membrane and ossicles, and that bone conduction was due to what was termed cranio-ossicular transmission. That this theory is erroneous is proved by the fact that bone conduction is increased, if altered at all, after the mastoidotympanal exenteration.

‡Eitelberg[8] showed that the normal duration of perception for the C^2 (512 d. v. s.) tuning fork on the various parts of the cranium was as follows: Mastoid, 30 seconds; forehead, 18 seconds; occiput, 20 seconds; teeth, 15 seconds. The normal duration of the same fork by air conduction being 58 seconds.

A tuning fork placed on any part of the cranium imparts its vibrations to the bone, and these vibrations are distributed to all parts of the head. Why then may we not consider hearing by bone conduction as simply the result of "sympathetic vibration," especially since Shambaugh's investigations have anatomically enhanced the Helmholtz theory of hearing? We know that we can create vibrations in a stable tuning fork by bringing a vibrating one of its own pitch in proximity or by touching a stand on which the quiet fork rests with vibrating fork. These induced vibrations will be of less intensity than those of the exciting force and, consequently, shortened in duration or perception, for there is a leakage of energy through friction or wave emission, so that the forced vibrations only increase up to the point at which leakage of energy balances the energy put in by the applied force; therefore, in resonance there is always a loss of energy, and the amplitude only increases until the loss is balanced by the gain from the work done by the applied force. If there were no dissipation of energy, the vibrations would increase indefinitely when the periods coincided. Why then may we not postulate the following explanation of bone conduction?

With a vibrating tuning fork held on the mastoid, or on any part of the cranium, we hear its vibrations through the creation of "sympathetic vibration" in that area of the tectorial membrane tuned to respond to the pitch of the fork used. The sound is reduced in intensity, and, therefore, in duration of perception because the tectorial membrane can only vibrate up to a point where the leakage of energy balances the energy put in by the fork on the mastoid.

SUMMARY.

The whole matter may be summarized as follows:

1. By both air and bone conduction it is necessary that we have some index which will indicate the intensity under which a tuning fork is heard, and the simplest way to obtain a factor of this description is by comparing the abnormal duration of

perception with the normal duration of perception and taking the difference betwen the two as the significant indicator.

2. As the energy necessary to make a tuning fork heard by bone conduction is markedly greater than the energy necessary by air conduction, the normal duration of perception of each is different; therefore, we may not directly compare the one with the other, but we must make separate tests of each course of sound conveyance.

V.

The tuning fork tests being wholly and purely subjective, we have been led to doubt, not only the results of others, but our own findings as well, and for this reason so many qualifications and exceptions have crept into acceptance that each individual seems to have a system of his own, and the original tests are almost worse than useless. If we can bring ourselves to postulate dogmatically one rule, and working from that prove or disprove the whole, our results will assume a different aspect. Accepting resonance as the chief factor governing bone conduction, I consider that a shortening of ten seconds or more by bone conduction is abnormal, especially if it is unilateral, the other ear being normal, and if contraaudition has been prevented by the use of the "noise apparatus."

The older investigators were hampered and confused by the impossibility of excluding one ear while the other was being tested, as the occurrence of contraaudition, the bone conduction from the mastoid of one ear to the opposite ear, prevented a true determination of the duration of perception by bone conduction, and this was the reason so many restrictions were placed on the use of the Rinné test by such scientists as Hartman, Bezold, Lucae and others, and the discrediting of the Schwabach test. The introduction by Bárány of the noise apparatus has made it possible to diagnose total unilateral deafness, and, what is more important, to determine the duration of perception of the sound of a tuning fork by bone conduction in each ear separately. The use of the noise apparatus and the possibilities of original work along this line has not, to my mind, been sufficiently investigated. We are inclined to think that the instrument is only of value to exclude one ear in testing for total deafness. As a matter of fact it should be used whenever bone conduction is tested, and there is no reason why we may not be able to test restricted areas of the tectorial membrane by the use of a scientifically constructed suppression apparatus.*

VI.

In the use of the tuning forks of different pitch it must be remembered that the amplitude of the forks of low pitch is wide, the time rate of decrement slow, and the intensity weak; therefore, a slightly defective conduction apparatus will fail to convey these vibrations when the normal ear will still transmit and perceive them for a long time afterwards. With the forks of high pitch the amplitude of the vibrations is short, the time rate of decrement very rapid, and the intensity very strong; hence, the difference between defective perception and the normal perception of the duration of the vibrations may be so short that it is immeasurable. (Figure 3.)

By the use of three tuning forks, the C (128 d. v. s.), the C^2 (512 d. v. s.), and the C^5 (4096 d. v. s.), the useful range of hearing may be covered, and my routine examination is made in the following order and manner:

(1) The Weber test.

(2) The Schwabach test, the duration of the perception by bone conduction for the C^2 fork as compared to the normal. The noise apparatus tuned to cut out this fork is used to exclude the ear not being tested.

(3) The duration of perception for the C^2 fork by air conduction as compared to the normal. (The method to be described later.)

(4) The duration of perception for the C fork by air conduction as compared to the normal. (The method to be described later.)

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*Francis R. White exhibited before the Ninth International Otological Congress, Boston, 1912, a noise apparatus which is simple and inexpensive, and yet is the most useful and adaptable of all these instruments whose requirements he summed up as follows:

"(1) The sound must effectually exclude all other sounds.

"(2) The sound must not be transmitted to the other ear.

"(3) The sound apparatus must be regulated for varied hearing.

"(4) The use of the apparatus must not be disagreeable to the person being examined."

The White and the Neumann instrument fulfill all these requirements. The Bárány instrument cannot be regulated, and is disagreeably loud.

(5) The duration of perception for the C^5 fork by air conduction as compared to the normal. The noise apparatus tuned to great intensity is used to exclude the ear not being tested.

All these trials are timed with a stop watch, and the result of each is written in the form of a fraction the numerator of which denotes the patient's hearing and the denominator my own hearing. If there is a difference in our hearing in any test it is noted minus or plus, and this is the important factor that indicates the defect in the patient's hearing.

The Weber test, when properly explained to the patient, is usually accurate; it may be verified with the noise apparatus, but when the fork is not heard from the forehead or the teeth it is indicative of perception deafness. The Schwabach test should be made in the original way, by transferring the fork from the patient's mastoid to one's own, or vice versa.* The normal perception duration of a tuning fork is much more constant and much easier for the patient to determine by bone conduction than by air conduction, and the noise apparatus increases the ability to say when the fork is no longer heard. In estimating the duration of perception by air conduction it is not always easy for the patient or the examiner to determine when the vibrations can no longer be perceived; this is probably due to interference with the sound waves, reflections, etc., unless the fork is held in exactly the right position in front of the auricle. To avoid these factors I make use of a small stethoscope (Figure 4) upon which I rest the handle of the tuning fork, one tube being in the patient's ear the other in my own. The method is good for any fork below the C^3 (1024 d. v. s.), and has been advised in various forms by several aurists. For the routine bone conduction test the C^2 (512 d. v. s.) has proven the most valuable in my hands; it is heard and not felt, and it tests the central portion of the tectorial membrane. I look upon the tests with the various forks named as giving me a "field of hearing" the variations of which furnish the basis of differential diagnosis.

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*Years ago Kayser used a tuning fork mounted in the middle of a wooden bar, one end of which was placed on the patient's mastoid, the other on his own. Bárány suggests the use of an otoscopic tube. I find by this method that the physician's duration of perception is less than normal and that the tube creates a Weber reaction in the patient's ear. Wanner proved that the electrically stimulated forks were unnecessary, and that the test could be made as accurately by the older method.

The classification to be given presupposes that bone conduction when properly estimated is an infallible indication of the condition of the auditory nervous apparatus, that the C fork is the lowest in the key of C allowing an approximately accurate estimation of the intensity under which this note may be heard and not felt, and that the C^5 fork should be heard, under its minimum intensity, in all cases of conductive deafness.

VII.

(A) Conduction deafness, including affections of the eustachian tube, when:

(1) The Weber reaction is referred to the ear complained of or said to be the deafer.

(2) The bone conduction for the C^2 fork is equal to or greater than the normal in duration of perception.

(3) The air conduction for the C^2 fork is reduced in duration of perception as compared to the normal. (Rinné showing all deafness to be by air conduction.)

(4) The air conduction for the C fork is reduced in duration of perception as compared to the normal, and to a greater degree than for the C^2 fork.

(5) The air conduction for the C^5 fork is normal in duration of perception.

Case 1.—K. S. is a patient who has had the radical operation and retained good hearing, distinguishing the whisper at four and conversation at ten feet. Bone conduction for the C^2 fork + 5″ (40″/35″). Her duration of perception for the tuning forks as compared to the normal shows that her hearing is purely a question of faulty conduction.

C —49″ (16″/65″)
C^1—27″ (13″/40″)
C^2—30″ (30″/60″)
C^3—10″ (15″/25″)
C^4— 3″ (18″/21″)
C^5— 1″ (9″/10″)

If this patient is accepted as a control case, and I feel justified in presenting her record as such, the record of her hearing can be taken as a demonstration of the physical properties of audition which have heretofore been described. Theoretically this girl has a form of deafness which, if we had an instrument of absolute precision as to intensity, could be shown to be actually the same for all these forks. That is to say, that a reduction of forty-nine seconds for the C fork does not indicate any greater degree of deafness than a reduction of one second for the C^5.

The case has been given to show that in pure conduction deafness the results of the tuning fork examination will follow the physical laws governing the same. There are exceptions, but these exceptions are often important, especially in the diagnosis of otosclerosis. If the bone conduction is increased, normal or slightly decreased (five to ten seconds) in duration of perception for the C^2 tuning fork, with a decrease by air conduction for the same fork which is equal to or greater than the decrease in the duration of perception for the C fork, one should be guarded in prognosis, for with a normal drum head, a patent eustachian tube, and the complaint of tinnitus, one is probably dealing with a beginning ankylosis of the footplate of the stapes. The explanation of this defect in hearing has puzzled me until the appearance in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY for December, 1915, of an article by G. M. McBean, of Chicago, entitled "Theories Concerning Paracusis Willisii," in which this pertinent inquiry is advanced: "Is there any longer a sound conduction apparatus when its most important link, namely, the fenestrostapedial articulation, has become ossified? The sound conduction has then ceased to exist as a specialized mechanism."

In making air conduction tests by means of the stethoscopic method, the sound waves are conducted directly against the drum head without interference; therefore, the vibrations from the C fork probably act in the same way as the jolting under which paracusis Willisii becomes a symptom. With the C^2 fork this element of jolting is by no means so marked, and we must actually hear these vibrations through the action of the ossicles. When the footplate of the stapes is free there remains a flexibility which prevents the sensation of vibration (pallesthesia), though the vibrations may not be transposed and transmitted as well. When ankylosis begins, the ossicular chain takes on the characteristics of a solid connecting bar between the drum head and the cochlea, rather than a perfect conducting medium; therefore, the vibrations of the C fork are felt under the law of "deep sensibility," whereas the vibrations of the C^2 fork are not suited to reach the sensorium by this route, and we lose in duration of perception for the C^2 fork to a greater degree than for the C fork. As the ankylosis progresses the hearing tests take on the characteristics of a simple conduction deafness of marked degree.

Case 2.—Otosclerosis. Mrs. C., aged thirty-five years. Complains of deafness slow in onset but progressive in character. Drum heads and tubes normal.

	Right.	Weber not referred.	Left.	
B.C. −12" (20/30)	A.C. −74" (15/89)	C^2	−54" (35/89) A.C.	—N (30/30) B C.
	A.C. −39" (15/54)	C	−36" (25/61) A.C.	
	A.C. N. (9/9)	C^5	N. (10/10) A.C.	

Case 3.—Conduction deafness, right; labyrinthine deafness, left. Miss K. H., aged twenty-eight years. Complains of deafness in the right ear which has grown worse within a few weeks. Left ear deaf for years. History of many recurrent attacks of earache when a child.

	Right.	Weber referred to right.	Left	
B.C. −5" (20/25)	A.C. −28" (15/42)	C^2	(Neg. /50) A.C.	−22" (10/32) B.C.
	A.C. —45" (15/60)	C	(Neg. /55) A.C.	
	A.C. − 1" (9/10)	C^5	(Neg. /10) A.C.	

Ear drums: Right markedly retracted in anterior segment. Handle indrawn. Left retracted, but not so markedly as right; scarred. This patient had an acute eustachian catarrh in the right ear, which quickly responded to treatment. No attempt was made to treat the left ear.

If the "fields of hearing" are contrasted in these cases, it will be noted that although the deafness for the C fork is much greater in Case 3 than in Case 2, the deafness for the C^2 fork in Case 3 is also much less than in Case 2. In Case 2 the greater degree of deafness is for the C^2 fork. In Case 3 the deafness is more marked for the C fork. In Case 3 the left ear is, for all practical purposes, totally deaf. Made in the ordinary way, Rinné would have been called negative because contraaudition was marked. With the noise apparatus it was a simple matter to determine the nervous element in the case.

VIII.

(B) Perception deafness may be considered complete when the field of hearing is as follows, assuming that the lesion is unilateral:

(1) The Weber reaction is referred to the better ear.

(2) The bone conduction for the C^2 fork is negative, the good ear being suppressed with the noise apparatus.

(3) The air conduction for the C^2 fork is negative, the good ear being suppressed with the noise apparatus, and the C^2 fork, stimulated to its greatest intensity, is not heard by the stethoscope method.

(4) The air conduction for the C^2 fork is negative under the conditions just described (3).

(5) The air conduction for the C^5 fork is negative. With this fork it is particularly needful that the noise apparatus be used.

Robert Bing, in his most valuable "Compendium of Regional Diagnosis in Affections of the Brain and Spinal Cord (second edition, page 134), has this to say: "Between nerve deafness—namely, that due to lesions of the cochlea nerve or its supranuclear tracts and that due to lesion of percipient structure in the cochlea—no symptomatologic distinctions can be drawn. From the neurologic point of view, therefore, the regional diagnosis of lesions of the auditory mechanism must be regarded as wanting in precision." As a matter of fact, the question of differentiation, though presenting problems of baffling complexity, is by no means beyond the realm of possibility. Gradenigo, to whom we owe so much along the line of acoumetry, has shown that it is possible, and Hollinger,[9] among others, has reported most convincing cases.

Accepting the theory that tone analysis takes place in the labyrinth, and that the cochlea branch of the auditory nerve serves but to convey all tones to the higher centers, we may assume certain things:

Labyrinth—

(1) If the intralabyrinthine structures are completely involved, we will have total deafness, both by bone and air conduction.

(2) If the intralabyrinthine structures, excluding the nervous elements, are but partially involved, we will find deafness by bone and air conduction, but, as a rule, the degree of deaf-

ness will be greater by air conduction than by bone conduction.

Nerve—

(3) If the nerve itself is involved, the deafness will be total in high degree of neuritis, both by air and bone conduction.

(4) In partial acoustic neuritis the bone conduction will be reduced out of all proportion to the air conduction.

Total perception deafness must be either labyrinthine or nerve, but we should look upon certain labyrinthine forms as conductive perception deafness, remembering that there is a sound conduction function to the labyrinth, and that only actual nerve involvement, peripheral or distal, constitutes true perception deafness. A massive effusion into the labyrinth would cause a total labyrinthine deafness simply because sound vibrations could no longer be conveyed from the footplate of the stapes—the nerve, however, might remain perfectly normal. A partial effusion into the labyrinth might only involve the vestibule or the first turn of the cochlea: in such a case sound vibrations could not continue their course when heard by ossiculoperilymphic transmission; therefore, air conduction would show a greater reduction in duration of perception than bone conduction, though the latter would also be reduced. Bone conduction, we assume, is due to resonance created by the vibrations taken up by the skull, and, therefore, if the areas of the tectorial membrane which responds to a certain tone is not greatly involved, bone conduction will be better than air conduction. In other words, the vibrations by air conduction must make their way through an obstructed passage; by bone conduction they are directly received in an area specifically suited to respond by indirect transmission. In such a case the energy exerted by the vibrations upon the tectorial membrane is greater by bone conduction than by air conduction.

If the auditory nerve is involved and the labyrinthine structures are intact, the vibrations follow more closely the normal physical laws. That is to say, that the energy exerted by the vibrations is much greater by air conduction than by bone conduction. Vibrations conveyed by ossiculoperilymphic transmission would get through a partial "nerve block," whereas, the weaker vibrations of bone conduction, created by resonance, would have no effect; and so, bone conduction would be much reduced in duration of perception, whereas air conduction might be, in slight cases of nerve involvement, almost normal.

To attempt to draw hard and fast rules along this line is, of course, impossible; the exceptions occur more often than the rule. As a question for study, however, I make the following classifications in cases of partial perception deafness:

(C) Partial labyrinthine deafness.

(1) The Weber usually referred to the better ear. At times to the bad ear in cases showing but slight reduction in bone conduction.

(2) The bone conduction for the C^2 fork reduced at least ten seconds in duration of perception, usually from one-half to two-thirds the normal duration.

(3) The air conduction for the C^2 fork negative or markedly reduced in duration of perception.

(4) The air conduction for the C fork negative.

(5) The air conduction for the C^5 fork negative.

(D) Partial nerve deafness.

(1) The Weber referred to the better ear.

(2) The bone conduction for the C^2 fork greatly reduced or negative in duration of perception.

(3) The air conduction for the C^2 fork better in duration of perception, or reduced to the same extent as the bone conduction for the C^2 fork.

(4) The air conduction for the C fork of good perception duration in recent cases, reduced markedly in old cases, but never negative.

(5) The air conduction for the C^5 fork reduced in duration of perception, or negative.

The basis of such a classification is the study of forty cases* of perception deafness which I have been able to make in the last year in my ear clinic at the Baltimore Eye, Ear and Throat Hospital, and in the Department of Neuro-Otology, which through the interest of Dr. Harry Friedenwald and with the cooperation of Dr. A. C. Gillis, we have been able to establish at the Mercy Hospital. In a later communication we hope to be able to show the importance of the hearing tests in conjunction with the vestibular tests of Bárány. In this paper I shall only speak of the examination of the cochlea branch of the eighth nerve.

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*This number has been increased since the paper was written, without change in result.

Case 4.—Partial labyrinthine deafness. Wm. A. D., aged twenty-one years (B. E. E. T. H. 102,924), was examined May 20, 1916. He complained that he had been almost totally deaf since February. Gradual onset. The patient was so deaf that all questions had to be written.

Ear drums.—Right: Thickened and congested. Bulging in posterior portion. Middle ear seemed filled with thick secretion. Left: Presented the same appearance.

The patient had had no pain, no nausea, no vertigo. Denied lues. He had, however, been a patient at the Maryland Tuberculosis Sanatorium from January 3 to January 27, 1916, and was pronounced by Dr. V. C. Cullen "a progressive case of tuberculosis with an absolutely bad outlook." Dr. Hirshman, who examined the man in May, was of the same opinion. The hearing tests gave the following results:

HEARING TESTS BY AIR CONDUCTION.

The Weber was referred uncertainly to the right. The air conduction was negative for the C, the C^1, the C^2 and the C^3 tuning forks under their maximum intensity. The C^4 showed —22″ (3/25) in the right ear, but was negative /25″ in the left. The C^5 fork was negative /10″ in both ears. The Galton was heard at three turns in the right and at five turns in the left.

Here undoubtedly was a case of serous labyrinthitis of tubercular origin. The deafness was of the conduction type, but labyrinthine as well as middle ear. The bone conduction tests are of great interest and were as follows:

HEARING TESTS BY BONE CONDUCTION.

Right.		Left.
Negative /35	C^2	—25″ (5/30)
—8″ (7/15)	C^1	— 6″ (9/15)

A paracentesis was done on both drums, which was followed by a thick, nonpurulent discharge that continued for some weeks. On June 7, 1916, his bone conduction had improved for the C^2 fork, and he could hear the C fork for a few seconds by air conduction.

Right.		Left.
B.C. —23″ (7/30)	C^2	—18″ (12/30) B.C.
A.C. —84″ (6/90)	C	—78″ (12/90) A.C.

The discharge ceased and the drums resumed a partially normal appearance, and by October 9, 1916, he could hear a loud conversational tone. The bone conduction for the C^2 fork had increased to 35/35 in both ears. The air conduction for the C fork had increased to right, —50″ (25/75); left, —30″ (30/60). He still could not hear the C^5 in either ear, and this was interesting from the standpoint that although bone conduction was normal for the C^2 fork, the C^3 and C^4 showed a decrease of —20″ and —18″ respectively. The tests of the vestibular apparatus, both by rotation and the caloric reaction, were of interest, and confirmed the labyrinthine affection. It is not the purpose of this paper, however, to go into that side of the question. The man's blood showed a negative Wassermann on two examinations.

Concerning tuberculosis of the middle ear, H. B. Graham[10] has this to say: "In functional testing of cases of tuberculosis of the middle ear, one is nearly always struck with the disharmony between the remarkable deafness and the length of time and the severity of the attack. The reduced perception for the voice and the low tone is, of course, the most marked factor, but the perception quantitatively for all tuning forks is also markedly and rapidly reduced, so that one is reminded of the luetic cases he sees following salvarsan, suggesting that there may be some similar toxic effect in the labyrinth as well as a disturbance of conduction. In the later stages a complete deafness may occur without vestibular symptoms having developed, and on sectioning the labyrinth we find a complete replacement of the labyrinth by a caseous organized mass." The case (Case 4) which I have just reported follows closely certain cases reported by Graham. One is borne out then by the pathologic findings in assuming from the hearing tests that the lesion is purely labyrinthine and that it is not a toxic neuritis in the true sense of the word. On the other hand, nerve deafness due to lues is truly an eighth-nerve involvement, and the results of the hearing tests in a case of partial perception deafness due to syphilis, when compared to labyrinthine case (Case 4) just quoted, will, I think, bear out the differential diagnostic points to which I have called attention.

Case 5.—L. S., aged forty-four years, was seen at the Baltimore Eye, Ear and Throat Hospital, June 12, 1916. He complained of poor vision and failing hearing; the patient thought this had been progressing for about six months. He admitted lues of twenty-five years' duration. Treatment only during the first six months following the chancre. To report his general condition as briefly as possible, the man is a tabetic with partial optic atrophy, the vision of the right eye being 18/30, of the left 18/50 at the first examination. His pupils are unequal, the left being the larger, and the response to light is sluggish. Ophthalmoscopically the optic discs show atrophy to the temporal side, more marked in the left eye. The visual fields are contracted for form and colors, and there is a small central scotoma for red in the left eye. His patellar reflexes are absent. His hearing tests with the tuning forks are as follows, his tympanic membranes being normal in appearance:

June 12, 1916.

Right.		<——Weber.		Left.
B.C. —5" (30/35)	A.C. — 2" (73/95)	C^2	—15" (47/62) A.C.	—25" (10/35) B.C.
	A.C. —10" (40/50)	C	—10" (50/60) A.C.	
	A.C. — 3" (7/10)	C^5	— 5" (5/10) A.C.	

Note that in the left ear the Rinné shows a marked reduction by bone conduction as compared to air conduction. That the C fork is practically normal in duration of perception, but that the C^5 fork shows a reduction of five seconds, which with this fork indicates a marked degree of deafness for a tone of this pitch. This is the characteristic hearing defect found in syphilitic cases.

In recent years there has been a considerable study of the syphilitic affections of the internal ear, and the chief diagnostic point brought out by all authors is that there is a shortening of the bone conduction out of all proportion to the shortening by air conduction, with but slight shortening, or loss, for the sound of the low forks and a tendency for the forks of high pitch to be reduced in duration of perception. One of the most interesting articles concerning this subject was presented by George H. Wilcut[11] at the San Francisco meeting of the American Medical Association in 1915. In this contribution Wilcut gives a good résumé of the subject to date, besides his own valuable observations, which include a far greater number of cases than had been studied by the majority of the authors quoted. The question of the pathology of the condition is somewhat uncertain, but the indications are that the lesion is most probably "a syphilitic infection of the nerve sheath."[12] Swift and Ellis[13] believe that syphilitic deafness should never be considered an isolated entity, but a manifestation of cerebrospinal syphilis. Whether or not there exists a syphilitic meningitis in all cases is yet to be determined, but certainly all observers agree that deafness in these cases is due to a neuritis of the eighth nerve, either toxic or caused by increased intracranial pressure.

Physically speaking, it does not seem possible that the reduction by bone conduction in hearing power could be out of all proportion to the reduction in hearing power by air conduction, for if the nerve is affected, how can sound vibrations be perceived, no matter how well they may be conveyed? This has already been explained. By bone conduction the energy necessary to create resonance in the tectorial membrane is much greater than the energy necessary in vibrations carried by air conduction; therefore, in these cases of auditory "nerve block," the weak stimulation produced by bone conduction resonance is not sufficient to get through, whereas the stimulation produced by the powerfully reinforced vibrations, received and converted into the form best suited by the drum head and the ossicular chain, is sufficient to overcome the block. Though not exactly comparable, a similar condition is seen in the reduced peripheral vision of optic neuritis, with the retention of almost normal central vision. In the Mercy Hospital Dispensary we have been able to follow a creditable number of cases of acoustic neuritis accompanying tabes, cerebrospinal syphilis and other syphilitic central nervous system lesions which we hope later will furnish observations worthy of report.

In conclusion, my apology for presenting many well known facts is simply that the question of perception deafness and the use of the tuning forks is, generally speaking, in rather a chaotic condition, regardless of the fact that many contributions to the subject are worthy of deep consideration. There is absolutely no standardization in the use of the tuning forks, each man using them as he pleases. Why should we persistently speak of "hearing" or "not hearing" a tuning fork, when a moment's thought will convince us that unless we know the intensity under which any sound is heard, its value as a measure of auditory power is useless? Why should we persist in the use of terms "negative" and "positive" Rinné, when we know that a "negative" is often found in perception deafness and a "positive" in conduction deafness? Why should we persist in comparing bone conduction and air conduction by a method that is most inaccurate, especially when by the use of the "noise apparatus" we may definitely determine the true bone conduction of each ear separately? And may not all these inaccuracies account for the report of but eight cases of internal ear disease out of seventeen hundred and fifty-three cases of ear disease (I take these figures from the report of one of the largest special hospitals in the South)? And yet no less an authority than Politzer says that the eighth nerve is oftener affected than any cranial nerve! I have no doubt that my paper is open to criticism. I make no claim for originality. Much that I have said has been said before, and better said. The work itself has required a great deal of patience, but as otologists we can do no better than to accept the opinion of Gradenigo: "That the proof based on the duration of perception of tuning fork sound furnishes, in pathologic cases, a deformed likeness of the hearing power; real progress of otology can only be reached when rigorous and exact methods in physical regard become established."

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13. Swift and Ellis: Jour. Amer. Med. Assoc., May 1, 1915

The bibliography of the subject is, of course, exceedingly extensive. The author has made no attempt to give exact references except when it seemed necessary. The object has been to gather together into a workable whole those facts which are far enough removed from the hypothetical to be practical.

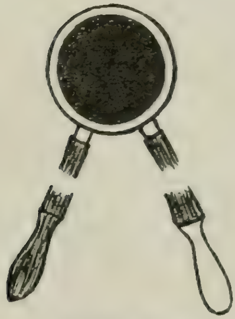
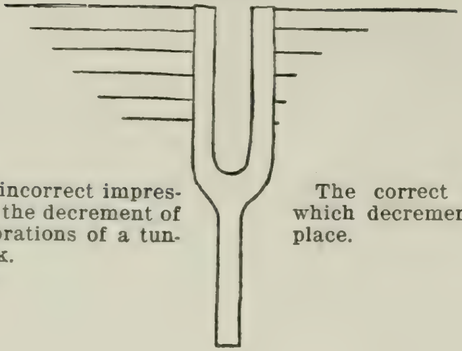


FIGURE 4.

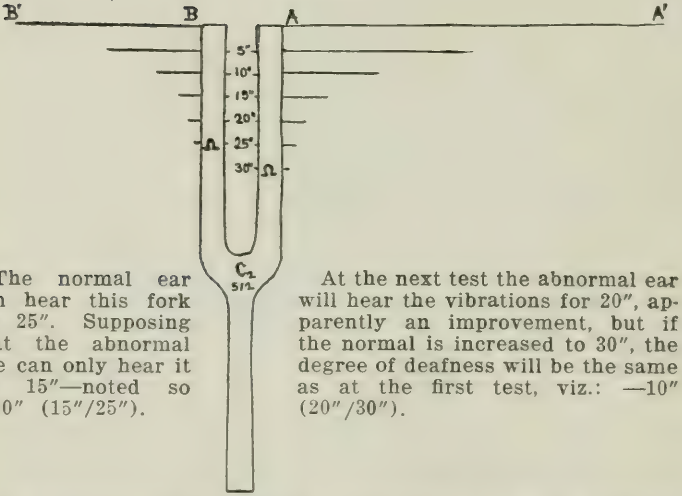
FIGURE 1.



The incorrect impression of the decrement of the vibrations of a tuning fork.

The correct way in which decrement takes place.

FIGURE 2.

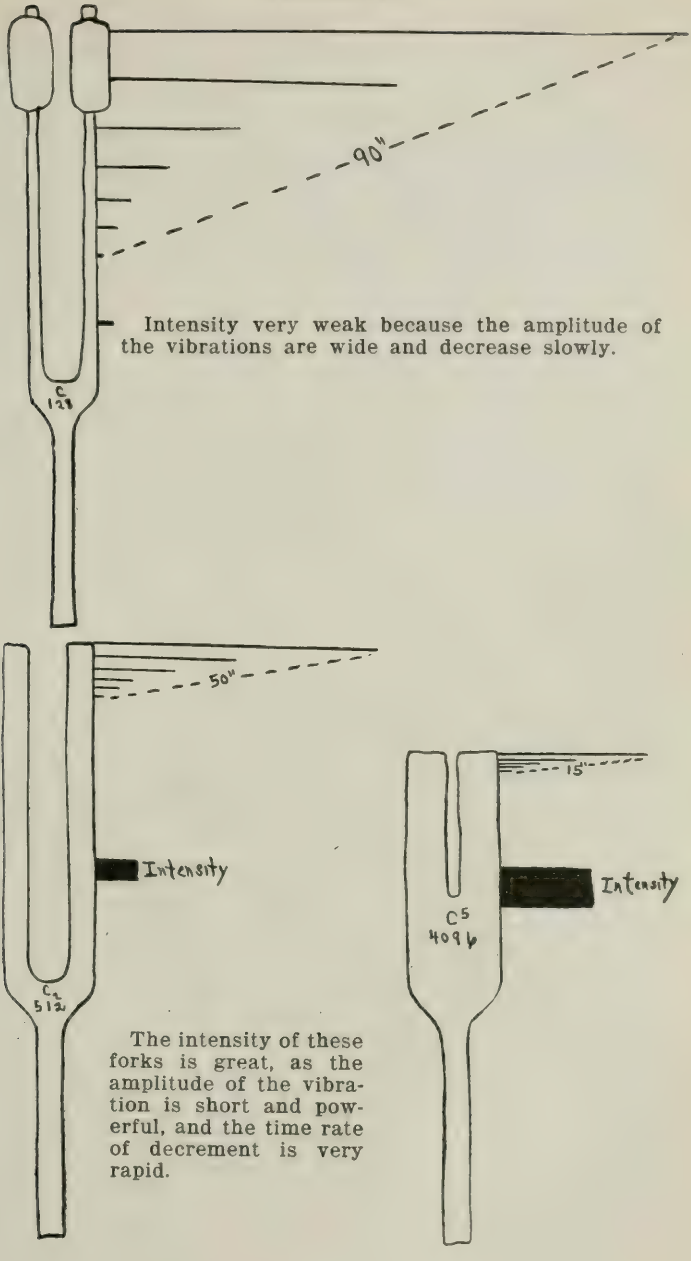


The normal ear can hear this fork for 25″. Supposing that the abnormal one can only hear it for 15″—noted so —10″ (15″/25″).

At the next test the abnormal ear will hear the vibrations for 20″, apparently an improvement, but if the normal is increased to 30″, the degree of deafness will be the same as at the first test, viz.: —10″ (20″/30″).

The duration of perception is immaterial. It is the difference between abnormal and normal perception which is the important factor.

FIGURE 3.



Intensity very weak because the amplitude of the vibrations are wide and decrease slowly.

The intensity of these forks is great, as the amplitude of the vibration is short and powerful, and the time rate of decrement is very rapid.

III.

SUPPURATIVE LABYRINTHITIS—A CRITICAL REVIEW OF ITS DIAGNOSIS AND TREATMENT.*

By Arthur B. Duel, M. D.,

New York.

Although the subject of orientation and equilibration, and the intimate connection of the vestibular apparatus with this function, have occupied the attention of otologists for more than a century, and, although much of the most important experimental work connected with it was done thirty or forty years ago, the surgery of the internal ear lesions resulting from inflammatory invasion has been developed well within the past two decades. I remember in the course of a summer in Berlin, about fifteen years ago, seeing the operation for suppurative labyrinthitis performed by Dr. Jansen on two chronic cases and one acute case. At that time the test for loss of hearing was very inadequate, and no one had suggested separate examination of the vestibular apparatuses. I recall that the operations impressed me with the importance of more careful anatomic study of the petrous bone with reference to operative interference, and that the technic of the operation interested me much more seriously than the diagnosis or the indications for operation.

It was not until Bárány, about ten years ago, presented the noise apparatus, which enables one quickly and positively to demonstrate complete loss of hearing, and the caloric reaction, which enables one to test separately the presence or absence of the static function, that surgery of the labyrinth was placed upon its present sound footing. Compared with all other means for determining the necessity of surgical interference in the internal ear, these two tests stand out in paramount importance. Indeed, in my own opinion, they are the only necessary functional tests. While the other functional tests, like whirling and the galvanic current, are interesting and useful in clarifying our minds regarding the static function, and therefore valuable in demonstrating certain reactions in the study of the subject, when we get down to an actual clinical case on which the question of surgical interference is to be settled, they are, to my mind, useless in comparison with the ones first mentioned. This is due to the fact that the whirling test can never cause a reaction in one vestibular apparatus at a time; and despite the fact that an imbalance of the two apparatuses may be demonstrated by the after-nystagmus resulting on turning first in one direction and then in the other, it is well known that compensation takes place sometimes in a longer, sometimes in a shorter, period after complete destruction of one side; and therefore a negative result will never be convincing. I believe it is absolutely useless in acute cases when a spontaneous nystagmus is present; and may be so in the chronic cases, owing to partial or complete compensation by the sound side. It seems useless in the acute cases, because with an imbalance sufficient to have caused the spontaneous nystagmus, the added confusion of the whirling brings out only phenomena which it is almost impossible to observe accurately. From another point of view, even if the information gained should be accurate, it seems hardly wise to jeopardize the chances of recovery of so ill a patient by subjecting him to this additional insult, when a much more positive test (the caloric) may be used with comparatively little discomfort. The whirling chair, in my opinion, then, should be relegated to the armamentarium of those who are teaching or studying the phenomena of static equilibrium, and should be discarded as a positive diagnostic factor in labyrinthine surgery. No one would think of not verifying its positive indications by the more accurate caloric test, and no one should accept its negative indications as being of any particular value.

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*Read at the meeting of the New England Otological and Laryngological Society, Boston, November 23, 1916.

The galvanic reaction, while it enables one to test each labyrinth separately, is confusing, from the fact that it may excite reactions by stimulating the vestibular nerve trunk, even when the end organ is not functionating. It is, therefore, in my opinion, useless except for the teacher or student, and is of no positive aid in determining the necessity for surgical interference, as compared with the caloric reaction.

These two tests stand in relation to the surgical diagnosis of labyrinthine involvement in about the same importance that transillumination occupies in the diagnosis of accessory sinuitis. Transillumination may be of positive value or it may not; in these days of radiography, no one would think of accepting its most positive indication without the confirmation of a radiogram.

The "fistula test" may be positive, in the absence of a positive caloric reaction, or it may be negative in presence of a positive caloric reaction. This, of course, will depend on whether there is a fistula present or not, and whether there is a vestibular function present or not; but one will seldom find a fistula test positive without also finding a caloric test positive, although it may be delayed or weak; while the fistula test, even in the presence of fistula, has no diagnostic value unless it is positive.

There is another important feature in connection with this test—and since I am on the subject, I may as well finish it now, although it may be somewhat out of place. The fistula symptom is present only in cases in which a suppurative process has extended to the membranous labyrinth, either by erosion of bone, by a necrotic process, or by extension through a dehiscence in the bony capsule. We seek for it in cases in which the symptoms point to a lesion which has not invaded the endolymph. This is in a type of cases (the socalled circumscribed) which often suddenly become active, acute, and develop into the most serious complete endolabyrinthine involvement. When we find a fistula during the course of an operation, every surgical instinct urges us not to probe or in any way exert the slightest pressure or traumatism on the fistula, and we so instruct our students. (This applies to cases where a functionating labyrinth has been demonstrated.) Under these circumstances, who can say that the fistula test once applied may not be the cause of converting a perilabyrinthitis into an acute diffuse suppurative one. The pneumatic pressure necessary to bring about the test cannot be calculated to fit all cases, and certainly the fistula test, applied as we usually see it, is quite as dangerous to the welfare of the patient as the probing or curetting during an operation—which we all abjure with such holy horror.

If the caloric test is positive, then, or can be demonstrated in even its weakest degree, one can well afford, in the interest of the patient, to wait and discover by inspection at the time of a radical operation whether or not a fistula is present. If the caloric test is negative—hearing also being entirely ablated—there might be a slight justification in applying a fistula test to determine whether or not there is any remaining vestibular reaction. With any hearing present, however, there would be sufficient contraindication to operative interference on the labyrinth to make the fistula test unnecessary, since at the time of the radical operation this might be determined by actual inspection. Here again, then, a test which is interesting and often very illuminating can only be practiced with certainly always a slight, and in many cases a grave, danger to the patient. If this test is used at all, it should be undertaken with great care, using the slightest pressure at first, and only increasing it on negative results up to a point (which must be guessed at) which the investigator thinks would not be sufficient to break through the delicate endolymphatic membrane. Having once demonstrated the presence of a fistula by this method, I consider it very bad practice to go on demonstrating it repeatedly, as we often see done for the illumination of one surgeon after another, in both hospital and private practice. The life of the patient with such a lesion is so seriously menaced by possible extension into fatal areas by the experiment, that one should—if he attempts it at all—record the result once for all.

This leads me into another digression. Much of the experimentation or demonstration of various interesting phenomena connected with an inflammatory involvement of the labyrinth might, in the interest of the patient, be well taken for granted. For example: as I have just said, if you will make use of a dangerous fistula test, you might at least be conservative enough, if positive, not to repeat it. If a patient suffering with an acute labyrinthitis has manifested symptoms which show that both the hearing and static sense are ablated, you may as well take it for granted that if made to stand he will fall toward the side of the lesion; that if whirled he will probably show certain reactions; that if moved about he will exhibit nausea and vomiting. Now, in most of the cases reported we see these results recorded. We all know that these phenomena would have occurred, and the man who has demonstrated them again has done so to the detriment of his patient; for we know that perfect quiet is absolutely essential to the prevention of extension from an endolabyrinthine lesion to the meninges, and may be so for the prevention of an extension from a peri- or paralabyrinthitis into the endolymph. To say in one breath that a patient should avoid undue excitement and any action which might jar the delicate barriers which nature is attempting to erect to prevent extension, and in the next breath to take such a patient out of bed to demonstrate that he will fall in a certain direction on attempting to stand or walk; or that he will show certain reactions to the insult of whirling him in a revolving chair; or even to stirring him about in bed or sitting him up in order to demonstrate a difference in the direction of nystagmic movements; or to provoke nausea and vomiting, is, to my mind, an exhibition of bad judgment or bad practice in the management of the case.

All these questions of whether the patient will fall in a given direction or react to certain experiments have been settled; we know they will do it. If that is unknown ground to any one, he should either accept it as a fact established by others, or learn it for himself on chronic cases, operated cases, and animals, rather than to demonstrate it by experimenting on his acute case, in which the demonstration may be the last straw which "breaks his camel's back."

This leads me to another somewhat critical position regarding the clinical diagnosis of labyrinthitis. We are deeply indebted to the Vienna school for their painstaking efforts to record the last detail of their cases, for their enthusiastic rivalry to present some new phase of an intricate subject, or some added factor in the differential diagnosis of the various types of labyrinthine inflammation. Their efforts to subdivide their cases and reduce each one to its lowest terms have been due in part, I think, to a desire to present a fixed or standardized formula, with definite symptoms for each and a definite treatment for each. This might, at first thought, seem to simplify the matter for students. I believe that instead of accomplishing this object, they have unnecessarily complicated the question, and have led those who approached their first cases of labyrinthitis into a feeling of bewilderment lest they should fall into a wrong diagnosis, on the one hand; or lest they should misinterpret the clinical symptoms and be led into performing an unwarranted operation, or be influenced in too long deferring a necessary one. The cases I am speaking of are all those which have resulted from a suppuration in the middle ear. The question which confronts one in such a case is: Is this acute or chronic? We know that no acute case of labyrinthitis can be present without manifest symptoms. We must remember that symptoms of an acute labyrinthitis may be present in either an acute or a chronic suppurative otitis. The first evidences of an acute inflammatory involvement of the labyrinth are impairment or loss of hearing and tinnitus aurium from involvement of the cochlear apparatus; and loss of equilibrium, vertigo, nausea and vomiting, and nystagmus resulting from loss or impairment of the vestibular apparatus. Either one or both of these impairments or loss of function may be present in any acute case. If either function is present in any degree, it is safe to say that an acute suppurative endolabyrinthitis is not present at that moment. If both functions are ablated the case may or may not be one of acute suppurative endolabyrinthitis. If the loss of function has been very rapid (within a few hours) following a virulent acute suppurative otitis, or following the fistula test, or any operative interference in a case in which there has been a perilabyrinthine inflammation, the chances are greatly in favor of the lesion being an acute suppurative endolabyrinthitis. If such is the case there will be no return of function, even though the case recover without operation. On the other hand, if the loss of function came on slowly, with irritative symptoms showing first for a number of hours, or days, or weeks, there may be an eventual complete loss of both functions, followed by final recovery or partial recovery of one or both, provided the labyrinth is not operated.

It can be readily seen from these incontrovertible facts that in any case of acute labyrinthitis we are brought face to face with the question of whether or not we shall operate upon a case which is in imminent danger of an intracranial involvement. The clinical symptoms from which we are able to elucidate the question may be indentical, and if all cases were operated upon to relieve such symptoms, there would undoubtedly be some which, had they been let alone, would have recovered with considerable function; on the other hand, some might be caught at the proper moment and drained with sufficient skill to prevent an intracranial involvement; and still again, there would undoubtedly be some cases in which the operative interference itself would precipitate the very cataclysm which the operator was endeavoring to avert. By this I mean that the excavation of bone necessary to accomplish the drainage—removed in the most ideal method imaginable—cannot help but be a menace to the delicate fabric which nature in every such case is attempting to erect between the meninges and the infected area. The thought of this great danger of traumatic production of the very mischief one is trying to avert has been growing upon me for years, and although I have carefully avoided employing mallet and chisel—the use of which I consider extremely bad technic on account of the violent concussion—I still feel that in these acute cases even the most careful excavation by the use of rongeurs, curettes and drills will in a slight degree present the same possible danger.

Under these circumstances I am strongly of the opinion that any acute labyrinthitis showing no symptoms outside of the labyrinth stands a better chance of recovery unoperated until the acute symptoms have subsided. This may mean—depending upon the condition of the mastoid—a few days or a few months, or for all time. During this stage of acute symptoms the endeavor should be to secure complete rest for the patient. If there were a question of whether I would operate upon such a patient or not operate, and in the meantime subject him to the insults to which he is usually subjected, I would say by all means operate. I mean by this that to take such a patient and stand him up or ask him to endeavor to walk to note which way he falls; or have him sit up in bed and put him through hot and cold water irrigations; or put him in a whirling chair and revolve him several times in each direction; or in any way disturb him so as to provoke the violent vomiting which such patients are likely to have at that time, is quite as likely to add the traumatic shock which might break down the barrier to the meninges, as a carefully wrought operation, but without presenting him with the advantage which the drainage afforded by the operation would provide.

Now, what symptoms are necessary to hold us in this waiting position?

Just two. (1) Loss of hearing. How is this demonstrated? With a suitable noise apparatus shutting out the sound side, the patient is unable to hear shouted words or a Galton whistle. (2) He has a nystagmus of the vestibular type, the slow movement of which is toward the diseased ear. (I shall speak more at length on this symptom a few moments later.) It does not matter whether the nystagmus is horizontal or rotary in character; you simply wish to know if a nystagmus characterized by a slow movement in one direction is followed by a rapid movement in the other direction. If the slow movement is toward the diseased side (it is almost sure to be if the hearing is ablated), it is the result of a great impairment or a complete loss of function on the diseased side. It is almost always rotary, or combined rotary and horizontal; but it is quite unnecessary to be too accurate about whether one or both are present, so long as there is a characteristic slow movement toward the diseased side, followed by a rapid movement in the opposite plane toward the other side. If once or twice in your lifetime you see the slow movement away from the diseased side, with quick recovery toward it, you may be certain that the labyrinth is functionating; that the nystagmus is the result of stimulation; and that the case has no question of a possible operation in sight, because there can be no endolabyrinthitis present. In such a case there would also almost certainly be some hearing present.

We all know that the acute symptoms in such cases rapidly subside. The function—hearing, static sense—may partially recover, entirely recover, or be completely lost. This applies to one or both. In the meantime a condition will be left in the labyrinth, depending upon what sort of resolution takes place. Of course, the cases which recovered function did not have a suppurative invasion of the endolymph. It is even possible that some of the cases which recover with loss of function may not have had a suppurative endolabyrinthitis. This is drawing pretty fine, however, as all of them have had an inflammatory invasion of the labyrinthine walls sufficient to upset or completely destroy the function of the delicate end organs in the cochlear and vestibular apparatus. The distinction only indicates how imminent meningeal involvement had been. There is no way of knowing which is present at the time. The case will either have recovered with or without function, or will have rapidly passed into what, for all practical purposes, may be considered a chronic labyrinthitis.

What symptoms during this anxious period of waiting should lead one to operate?

First, a temperature over one hundred degrees, accompanied by headache, photophobia, exaggerated reflexes, a positive Kernig, might be an indication that a beginning meningeal irritation was taking place, and no one should hesitate for a moment to operate, not only draining the labyrinth spaces, but also uncovering and incising the dura as nearly as possible at the internal auditory meatus and possibly over the temporosphenoidal lobe as well. This operation would be done, not for the labyrinthitis, but for a beginning meningitis which had not been averted by jealous care and watchfulness.

Another situation might be present which would present some of these symptoms and which might justify an operation in the midst of an acute labyrinthitis, but which might not demand an invasion of the labyrinth. This situation is more likely to arise in an acute labyrinthitis coming rapidly on top of an acute otitis before or without any clinical evidence of involvement of the mastoid. Such a case might be running a high temperature, owing to an acute follicular tonsillitis or an acute nasopharyngitis, or from an acute suppuration of one or several nasal accessory sinuses, the direct infection of the middle ear having occurred in the midst of their acute symptoms. Headache might be present from high temperature;

there might be some question of stiffness of the neck from glandular infection; there might be a questionable or very positive evidence of an acute mastoiditis or a sigmoid sinuitis. In such a case I believe one is warranted in operating the mastoid with as little concussion as possible in the presence of an acute labyrinthitis, being guided at the time of operation as to whether or not the labyrinth should be drained, by a spinal puncture and examination of the fluid for symptoms of meningitis. This can be done, where laboratory facilities are easily available, without any delay, the report being returned to the operator long before the mastoid excavation has been completed.

In the absence of evidence in the cerebrospinal fluid of a beginning meningitis, I should, even under these circumstances, advise leaving the labyrinth alone. I am quite aware that this practice is considered too hazardous by many, but my own experience and that of others with whom I have seen cases, has been fortunate enough to warrant that conclusion—for the present at least.

Reverting for a moment to the subject of spontaneous nystagmus (one of the manifest symptoms of acute labyrinthitis), you will notice that I spoke always of the slow movement of the nystagmus first, and always of the slow movement as one of diagnostic importance. You are aware that all nystagmus of a vestibular origin is characterized by a slow movement in one direction, followed by a quick recovery movement in the opposite direction. You are also aware that the slow movement is the only one for which the vestibular sense is responsible. It is a very good thing also to keep constanly in mind that the slow vestibular movement is always away from the side which is exerting the most powerful influence. The vestibular impulses which keep us constantly informed of our position in space and influence our vision in fixing on moving objects, or on stationary objects whilst we are moving, might, for purposes of demonstration, be compared to a billard ball held in a stationary position by three rubber fingers on either side, by an equal pressure from three different angles. You can well imagine that the least let-up in tension on any of the fingers would mean that the stronger pressure from its opposing finger would move the ball toward the weaker side. Now a labyrinthine stimulation might increase the impulses on that side to make a stronger "push" than that of the opposite normal side; the eyes would then rotate slowly toward the normal side, making a rapid recovery movement toward the stimulated side. This may happen in the very earliest stages of labyrinthine involvement. It may be induced by hot irrigations.

In an involvement of the labyrinth which in the least impairs or destroys the function of the vestibular apparatus, the impulses from that side are weakened or ablated entirely. Going back to our billiard ball, the normal impulses from the sound side will push it toward the weakened one. The vestibular movement of the eyes (the slow movement of any vestibular nystagmus), then is invariably away from the side sending out stronger impulses, so that it follows that a slow movement away from a diseased ear must mean increased functionation of an overstimulated vestibular apparatus.

On the other hand, a slow movement of the eyes (of vestibular origin) toward a diseased ear, cannot be anything but the result of a weakened or absent function on that side. The normal impulse of the sound ear has produced this imbalance.* Suppose that we imagine another influence, quite outside of the little rubber fingers, which intermittently, violently and rapidly jerks the ball back to its original position by a much more powerful impulse than they exert. This would typify a central influence on the eye acting from a center quite separate from the vestibular apparatus, which in vestibular imbalance is entirely designed as a compensatory act. Why under such circumstances have we fallen into the habit of naming the nystagmus from this quick compensatory movement, instead of from the slow movement, absolutely of vestibular origin? Even if it does not upset our train of thought in observing any case with labyrinthine symptoms, it only fails to do so because we transpose our terms. We cannot think of it properly without transposition; therefore, why should we not at least cease designating nystagmus by the direction of the quick recovery movement?

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*Note.—I wish to make it quite plain that I am speaking of vestibular lesions only. It is well known that a cerebellar lesion would produce a nystagmus in just the opposite direction. Destruction of a labyrinth would induce a nystagmus, the slow movement of which would be toward the destroyed side. Subsequent involvement of the cerebellum on that side would reverse the nystagmus. There are many differentiating phenomena brought out by pointing tests, direction of falling, etc., a discussion of which is quite beyond the limits of this paper.

We might say, in describing symptoms, that a nystagmus of vestibular character is present. This would mean that there is a slow and a quick movement; if we wished to be logical, we might say the slow movement (the vestibular one) is away from or toward the diseased ear, as the case might be. Without transposition of terms the beginner, and the experienced diagnostician also, for that matter, might then have a definite notion of what was happening in the vestibular apparatus.

We have thus far said nothing about the diagnosis or treatment of a chronic case. All chronic cases are characterized by the fact that there are no gross manifest symptoms. We discover indications of a chronic labyrinthitis in the course of a functional examination of a case of suppurative otitis which has formerly passed through an acute labyrinthitis with destruction of both hearing and static sense. The clinical history of a previous train of symptoms similar to those just recorded in the acute case might lead us to expect to find such a lesion. The demonstration of the presence of a dead labyrinth is perfectly simple. The functional test of hearing will show its complete ablation. The caloric test will show complete loss of function on the diseased side. You will remember that for practical purposes I included as chronic cases all acute cases which had ceased to exhibit spontaneous evidence of labyrinthine involvement. There is no need whatever of hesitating to employ for purposes of experimentation any of the tests which were so severely criticised in the investigation of acute cases. The rotation test may demonstrate a very marked imbalance in the vestibular apparatuses, the after-nystagmus resulting from rotation in one direction being much more marked than that in the other. If this is the case, standing with the eyes closed; walking forward or backward with the eyes closed; walking forward and suddenly looking in either direction; attempting to do things in the dark which formerly could be easily done; standing on one leg; walking or standing on an inclined plane, will all, in various degrees, show an unstable equilibrium, amounting in some cases to a complete loss. If he is whirled and his head placed in one position, he will fall in a certain direction; and if whirled and the head placed in another position, he will fall in another direction. The extent of his response to these experiments will depend upon the rapidity with which he has compensated for the loss of function of one of his vestibular apparatuses.

In a general way the length of time following the complete destruction will have much to do with his responses to these experiments. Nevertheless, just as one person may learn to walk a narrow plank or do some unusual athletic feat very quickly, while another may take a long time to do it, or never be able to do it at all, so we may measure the variation in compensation for the loss of one static apparatus that we may find in any such case. It follows, then, that after all, no matter how many experiments we may try from an interest in the phenomena, we are led always to the confirmation of any evidence we may derive from them by the result of the caloric reaction. If there is any remaining function in either the cochlear or the vestibular apparatus, an operation for drainage of the labyrinth is unjustifiable.

For practical purposes, therefore, if we have neither the time, nor the apparatus, nor the inclination, it is not necessary to do more than make the functional tests of hearing and the caloric test for vestibular reaction, in order to determine, so far as the functional test goes, whether or not a chronic labyrinthitis should be subjected to operation. To my mind, a fistula test in these cases is fraught with quite as much danger as in the acute cases, because one never knows how large the fistula may be or how insecure the barrier which nature has erected between the meninges and the suppurating focus.

It has been stated by a man of wide experience, and reiterated by many who have followed him, that complete compensation for loss of function on one side is a contraindication to invasion of the labyrinth on the dead side while doing the radical mastoid, on the ground that in such cases the labyrinth had been healed by the deposition of bony or fibrous tissue in place of the former purulent material. This seems an irrational conclusion, inasmuch as I have operated on several cases in which the whirling test showed almost perfect compensation, yet which at operation revealed necrosis of the labyrinthine wall and purulent foci in both the cochlea and vestibule.

The fiat has gone forth from another man of large experience, and has been widely accepted, that no radical operation should be performed on a chronic suppurating ear, in which a dead labyrinth has been demonstrated by functional tests without opening the labyrinth at the same time. This also I think is an unwarranted conclusion, inasmuch as a radical operation on such a case of very long standing seems justifiable without invading the labyrinth, unless a very definite lead into the labyrinth is revealed on careful inspection. This is well demonstrated by the conclusion just mentioned by the other man of wide experience. From my own experience, I would say that no radical operation should be done on an ear showing a dead labyrinth without the intention of entering the labyrinth if careful inspection showed any lead in that direction. The reason why this fiat went forth was, I believe, because so many such cases operated, without drainage of the labyrinth at the same operation, resulted in the conversion of a latent focus in the labyrinth into an acute meningitis. The cause for such a catastrophe could only be attributed to a traumatic rupture of barriers which had been walling off the purulent focus from the meninges. This only emphasizes the position of those who insist that operation in such cases should be done with extreme caution for the avoidance of concussion or rough manipulation of any area which might possibly be leading into the labyrinth, unless such manipulation was to be immediately followed by free opening into both vestibule and cochlea.

Those who will operate with such care that they feel certain they have not broken down barriers which have held for months or years, may, if they find no lead into the labyrinth, safely omit drainage of the labyrinth with the expectation that the majority of such cases will recover without it. Occasionally a case may require later a labyrinthine exenteration, owing to the fact that latent foci of infection were present which did not show sufficiently gross evidence on inspection to lead the operator to enter it at the first operation. It is my conviction that this practice would avoid the necessity of the more extensive operation in many instances, and would not subject the patient to added danger in those cases that may require the second operation.

A word as to the kind of operation advisable in these different cases.

Inasmuch as no acute case is operated unless early symptoms of meningeal involvement are present, it follows that any operation is inadequate which falls short of draining the dura at the same time that the labyrinth is opened. Such cases should have the vestibule opened both in front and behind the facial nerve, the cochlea uncapped, and the scala and modiolus entirely removed so that the meningeal fluid washes freely through. The plate from the lateral sinus to the petrous pyramid should be entirely removed, a slit made in the dura as near the internal auditory meatus as possible, and drainage of the meninges at that point facilitated by a ribbon of rubber tissue inserted through the slit. A similar drainage of the temporosphenoidal region should be made, if there is evidence of rapidly extending meningitis.

Any chronic case exhibiting evidence of meningeal involvement should have a similar operation. The chronic case, however, which exhibits only a suppurating labyrinth, should be opened in front and behind into the vestibule, the cochlea uncapped with extreme care, to avoid breaking the modiolus down to a point which will open a communication with the meninges. If the granulating suppurating cavities are opened and washed out, they will heal rapidly. It is quite unadvisable, in my opinion, to curette granulations from these cavities, for fear of opening up an avenue of infection either through the aquaeductus cochlea or the aquaeductus vestibuli. It is advisable to avoid removal of these protecting granulations, just as we now avoid curetting the granulations in epidural or perisinus abscesses. Free exposure, washing and adequate drainage will be more successful. The removal of the inner plate from the sinus to the petrous pyramid in these cases, without any meningeal symptoms, prolongs the operation and increases the danger, without in any way aiding the recovery of the lesion.

I have said nothing definite about the classification of inflammatory conditions of the labyrinth, except to hint at the idea that it had been made too complicated. You will note that I have only mentioned acute cases in which the endolymph was invaded, and those which gave active symptoms from the perilabyrinthine inflammation. I have no idea of insisting that the classification into circumscribed labyrinthitis, diffuse serous secondary labyrinthitis, diffuse purulent manifest labyrinthitis, and diffuse purulent latent labyrinthitis should not be adopted if you wish to do it, but I do insist, since they all arise from a suppurative otitis, that you will be unable to differentiate between the diffuse serous secondary and the diffuse purulent manifest labyrinthitis in the majority of cases, except by time and the outcome regarding function; and that having made this distinction, even on that evidence, you may often still be wrong.

Dr. John B. Rae read a very excellent paper on "The Diagnosis of Inflammatory Diseases of the Labyrinth" before the Medical Society of the State of New York, at Saratoga Springs May 16, 1916, in which he simplified the classification into: (1) Acute diffuse labyrinthitis; (2) chronic diffuse labyrinthitis; (3) paralabyrinthitis—(a) with fistula; (b) without demonstrable fistula. To my mind, this covers the ground quite sufficiently, and includes all the cases under the more confusing classification first mentioned. If it were to be put to a vote, I might offer the amendment that the conditions be called: (1) Acute endolabyrinthitis; (2) chronic endolabyrinthitis; (3) paralabyrinthitis—(a) with fistula, and (b) without demonstrable fistula; but I am not at all sure that the amendment might very justly be voted down in favor of the original proposition.

IV.

GUMMA IN FOSSA OF ROSENMUELLER CAUSING DEAFNESS.*

By Isaac M. Heller, M. D.,

New York.

Mrs. H., aged forty-one years, married, called October 25, 1916, complaining of deafness in the left ear. This had been growing progressively worse for three weeks, and the sense of fullness was most annoying. She denied all knowledge of any initial lesion, was the mother of three normal children, and aside of a seven months' stillborn child between the first and second children, there was nothing to suggest a luetic infection.

Examination of left ear showed a decided retraction of the drum, but no adhesions. Right ear, normal to hearing tests. Left ear, conversation, twelve feet; whisper, eight feet; acoumeter and watch, five feet; Weber to left; Rinné, positive; Schwabach, normal; large C and small C^4 forks heard, but not so well as on the right side.

The nose and mouth were negative. With the postnasal mirror, however, a mass was readily made out in the left side of the nasopharynx. It extended from the posterior wall forward to the palate, precluding a view of the lower and middle turbinates. At the site of the eustachian tube was a slight dimple or depression. Passing a catheter into this failed to inflate the ear. No undue force was used, as the diagnosis was not fixed, and I did not want to cause any trauma which might result in cicatricial contraction of the tubal orifice. This mass was about the size of a hickory nut. Surface was more or less covered with a false membrane, interspersed with smaller areas of reddish tissue. It seemed quite firm to touch of the catheter, did not bleed, and was not tender on pressure. Tumor was even better seen through the nasopharyngoscope of Holmes.

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*Read before the New York Academy of Medicine, Section on Otology, December 8, 1916.

The diagnosis lay between a malignant growth and a gumma, with a decided leaning toward the latter, despite the negative history. Tuberculosis was ruled out because of the single growth, lack of erosion, healthy apeparance of surrounding tissues, and absence of larynx and lung involvement. There was no glandular enlargement. A competent pathologist reported a four plus Wassermann.

On November 3d and 10th I injected neosalvarsan gram 0.45, and on November 23d, gram 0.60, intravenously. She was likewise put on a fifty per cent solution of sodium iodid with mercury inunctions. Reaction was prompt. After the first injection the mass was reduced by one-half; after the second, the tube could be catheterized, while after the third, hearing was equal to that of the right ear and Rosenmüller's fossa was free.

Examination of the literature shows this form of solitary tertiary lesion to be quite uncommon. Mucous patches of the secondary stage throughout the mouth and pharynx are frequently seen. Likewise a gumma of the soft palate with perforation and cicatricial contraction is often encountered. The interesting point in the case cited is the total freedom from symptoms referable to syphilis. If she had not noticed a disturbance in hearing, she would have sought no medical advice until sloughing of the palate had occurred.

Gerber reports a similar case, where, however, there was a clear history of primary infection some six years previous. In his atlas he shows an excellent reproduction of this condition, but there is an added involvement of the uvula. Gerber concludes by maintaining that tertiary syphilis can be present and concealed in the nasopharynx without any other manifestation of the disease elsewhere about the body. This is quite important to remember, since such a neglected case would surely be followed by ulceration of the soft palate or tubal opening, and more or less stenosis be the result. A small lesion is readily overlooked, especially where posterior rhinoscopy is difficult or impossible. It is here that the electric nasopharyngoscope of Holmes is of distinct value, and it should always be used where the otoscopic findings do not fully account for the disturbances of hearing.

A negative specific history should never mislead one in his conclusions, for with improved technic in serodiagnosis, cases of mild, overlooked syphilitic infection are yearly becoming more frequent. To illustrate, let me give briefly a hitherto unpublished case related to me by Dr. A. Rostenberg:

A married woman came to him with a diffuse papular eruption scattered about the body. It looked suspiciously like a secondary syphilide. Possibility of a primary lesion was denied. On close examination he discovered a scar the size of a dime on the sternum just below the clavicle. She stated this to be the site of a sore some five weeks before. He consulted the physician then in attendance, who said it was a hard indolent ulcer, yielding slowly to aseptic treatment, and lasted about four weeks. Because of its unusual location, he gave no thought to syphilis. Further investigation brought to light the fact there was a boarder in her house who had had a chancre, and that the patient took care of his linen. Contaminated finger nails and a scratch on the breast completed the chain of evidence. Subsequent Wassermann was strongly positive.

If this woman had disregarded her rash, which was of mild degree and apparently unimportant, she would have been totally innocent of the source of infection. Some years later, seeking relief from a tertiary lesion, she would have indignantly denied the possibility of lues.

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V.

A CONTRIBUTION TO THE HISTOPATHOLOGY OF THE NOSE AND THROAT.*

By Louis K. Guggenheim, M. D.,

St. Louis.

For many years the writer has been conscious of a certain feeling of shame in connection with his specialty: this feeling has resulted from the observation that whereas in practically all other fields of medicine, men who are preparing themselves for their life work begin with the fundamental studies of all branches of medicine, histology and histopathology, men who are fitting themselves for service to humanity as otolaryngologists usually begin with a mad desire to perform tonsillectomy, submucous resection of the septum, etc., and never do, with but few exceptions, apply themselves to the acquiring of the basic knowledge of their specialty, namely, the histology and histopathology of the nose, throat, and ear.

This neglect to build the foundation is probably more noticeable in Europe than here in America, where not so many men are found in one locality preparing themselves for a specialty. Practically all physicians who go to Europe to study, with the exception of otolaryngologists, devote much of their time to the study of pathology. The writer knows of many surgeons, for example, who have devoted practically all of their time in Europe to anatomy and pathology, and is unable to think of a single man, with the exception of otolaryngologists, among the hundreds whom he has met in Europe, who did not realize the necessity of fundamental work. How is it with the would-be otolaryngologist? Over to Europe for a few weeks or a few months to "learn" his specialty, he wants to know, fifteen minutes after his arrival in Berlin or Vienna, where he can get some operating, either on the cadaver or the living. Embryology, histology, histopathology! What can he do with such knowledge in his office when he returns? He smacks his lips over the fees he will demand for a radical mastoid, a septum operation, etc.

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*Thesis accepted by the American Laryngological, Rhinological and Otological Society.

No honest man can deny the fact that of all the specialties of medicine there are more superficial men practicing otolaryngology than any other branch of our science. Now, there is no argument whatever concerning the fact that even without knowledge of histology and histopathology one can get along in otolaryngology: one can even do much good in this world. I know men who remove turbinal hypertrophies, tonsils, and adenoids, who can perform a septum operation, simple and radical mastoid operations, etc., all very beautifully; men who have large incomes and help their patients, but how about progress in otolaryngology, and how about genuine satisfaction in one's work? These are impossible in the absence of a perfect understanding of the things with which one is dealing. Ah! yes, one hears, that is all very fine in theory, but practically why isn't it just as well to send the specimen to a pathologist for a diagnosis; and why bother about cells and groups of cells as long as one can recognize the macroscopic condition and treat it properly! I say such an attitude is disgraceful. In a specialty as limited as that of otolaryngology, it is a shame that men can be satisfied with a knowledge of the mere facts necessary for them to make a living and necessary to the carrying out of routine treatment and operations. Have we no pride in our special work? Have we no desire to compete with those in Europe who have worked out the problems about which we in America write so learnedly?

This contribution of microphotographs has been selected from a series of ninety-six recently taken by the writer, who is not a pathologist but an otolaryngologist, and invites criticism of his descriptions.

It is a significant fact that, although the books dealing with otolaryngology are numerous, but few contain more than a mere mention of the histology and histopathology of nose, throat, and ear; and even in those which contain splendid chapters upon pathology, we usually see sketches of specimens which may or may not be true to nature. Why so little use has been made of the microphtograph in otolaryngology is difficult to explain. Possibly the erroneous idea that microphotography is very difficult is the explanation. Microphotography is not difficult; a certain amount of technic must be acquired, of course; but this is possible for anyone with ordinary intelligence.

This contribution, the writer hopes, will serve to stimulate a deeper interest in the groundwork of laryngology, and serve as a plea for the more widespread use of this admirable method of illustrating papers and books.

There is an almost irresistible temptation to overstep the bounds of pathology in discussing each specimen, but, in a short work of this kind, one must confine himself strictly to the subject in hand.

TECHNIC.

The specimens were all fixed in four per cent formalin; then passed through the usual alcohol series, after being washed in water. They were then embedded in celloidin and sectioned in the usual way. Almost all the specimens were stained with hematoxylin eosin and mounted in the customary manner. Each specimen was carefully studied and the most interesting field selected for photographing. A Leitz microphotographic apparatus was employed. Most of the pictures were made with the first objective, a few with the second, but none with the oil immersion. An arc light is used in the Leitz apparatus for projecting the image. After focusing on a ground glass the light was modified with a ground glass and an amber glass. The exposures varied from five seconds to fifteen seconds, depending upon the thickness and coloring of the specimen. The developing and printing of the plates was carried out in the usual way, requiring very little more skill than is required to develop and print an ordinary photographic plate.

I.

CYLINDROMA OF NOSE.

Sarcomata sometimes contain masses of hyaline substance which is produced by the cells; or the cells become converted into this material; or the connective tissue and blood vessels are changed into hyaline material. The hyaline masses, which are observed in a great variety of form, force apart the cells. In mucoid and gelatinoid cancers the mucoid and gelatinoid masses develop within the nests of cells and give rise to very much the same appearance as described above in the sarcomata. Such a tumor may be called a carcinoma cylindromatosum.

In the microphotograph we see in the center an enormous triangular space, filled with a hyaline mass (2) and practically devoid of cellular elements. Grouped around this central hyaline mass are seen numerous smaller oval and round hyaline collections surrounded by sarcoma cells (1). That the cells have been crowded apart by the hyaline formation can be readily seen.

II.

CYLINDROMA OF NOSE.

In this picture, from the same specimen, are numerous irregularly shaped cylindromatous spaces. In this view one sees several small cylindromatous areas just forming. As in No. I, the sarcoma cells (2) are clearly visible.

III.

ANGIOMATOUS POLYP FROM SEPTUM.

Bleeding polypi of the septum usually spring from the mucosa of the cartilaginous septum, and are of rather frequent occurrence. The explanation of their frequent occurrence on the septum is the vascularity of that area which is known as the locus Kiesselbachi. The formation of the growth is

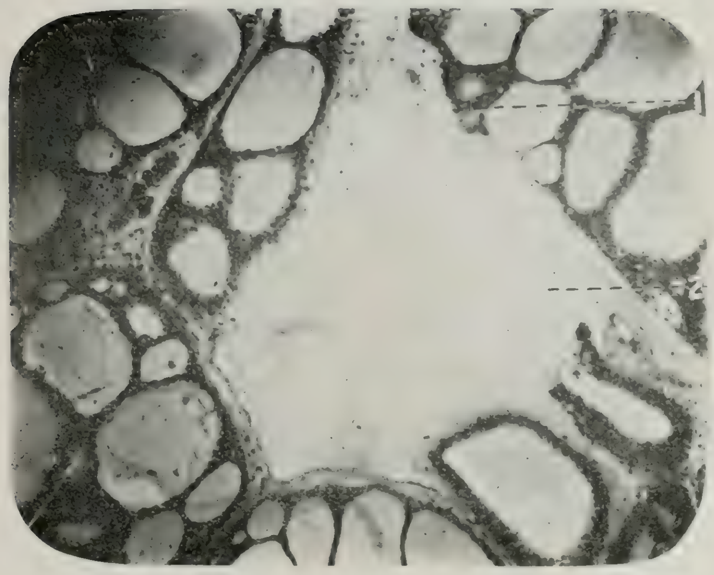

FIGURE 1.

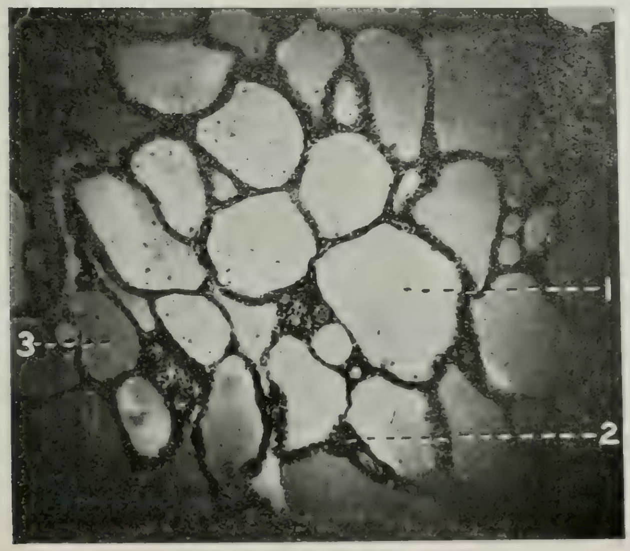

FIGURE 2.

made possible by a thinning of the mucosa with loss of elastic fibers; this permits of a proliferation and dilatation of blood vessels. This condition of the mucosa is probably the result of an inflammatory process. In the microphotograph are seen numerous, but for the most part comparatively small, blood vessels (3) lined with a single walled endothelium and showing no muscular coat. The blood vessels lie in a stroma of myxofibromatous tissue (2) containing many leucocytes (4). The epithelial covering (1) of the polyp is of the pseudostratified columnar variety.

IV.

CHONDROMA OF NOSE.

There is a difference of opinion among rhinologists as to the existence of a truly benign chondroma of the nose. It is probable that most of the cases reported as nasal chondroma are either ecchondromata (local hyperplasias of the septal cartilage) or chondrosarcomata. The writer believes that the specimen from which this microphotograph was made is one of benign chondroma of the nose, as there was no report of recurrence, and the growth was not the simple hyperplasia of the septal cartilage so frequently observed. A chondroma consists chiefly of cartilage. The amount of connective tissue found in its structure, covering its surface or penetrating its interior as a framework, is so slight that one almost fails to see it in many specimens. The usual tissue of a chondroma is hyaline cartilage; less frequently it is found to be composed of fibrocartilage.

The number, size, form and arrangement of the cells vary greatly in different tumors. Some contain many cells, others few; some have small cells and others large; others contain both large and small cells. The cells may have capsules or may not have; in some specimens they are seen to lie in groups surrounded by a capsule; in other cases the individual cells are found apart, but arranged in a regular manner. All varieties of cartilage which exist normally may be found in tumors; accordingly, cells of different forms are observed; the majority, however, are usually the round form. Commonly

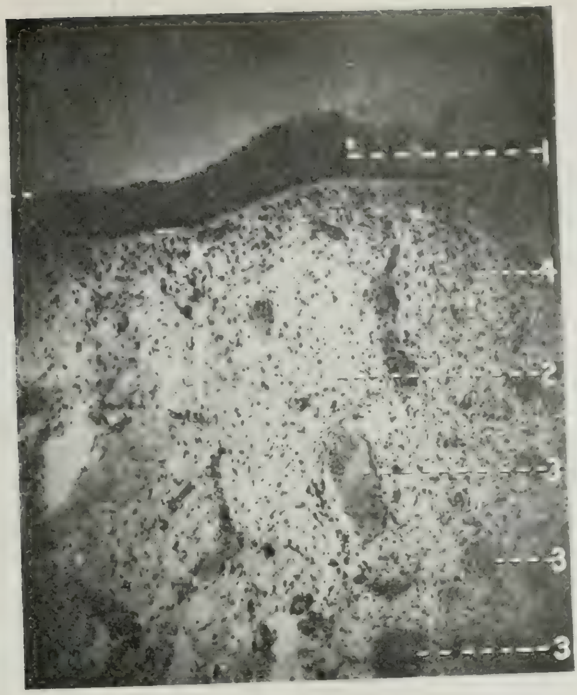

FIGURE 3.

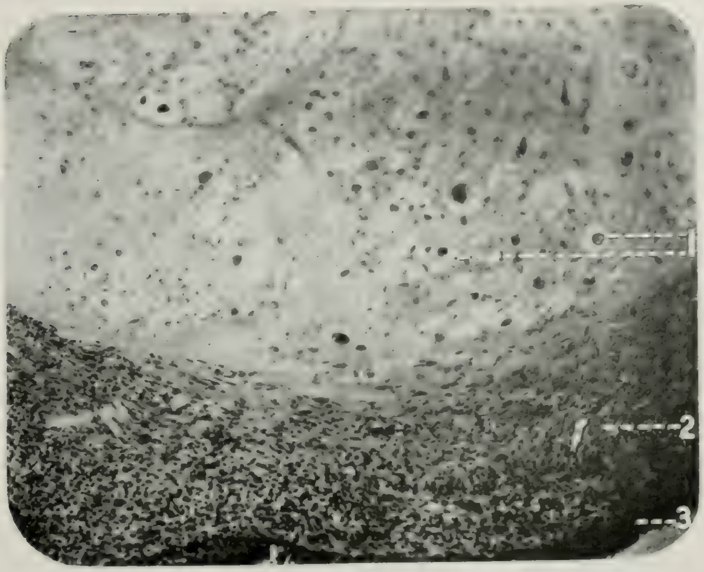

FIGURE 4.

spindle and star-shaped cells are found in the neighborhood of the connective tissue bands which separate the tumor into lobules or surround it as a whole. Retrograde metamorphoses are often observed in the chondromata. Fat drops are often seen in the cells. A mucoid degeneration of the basic substance is sometimes observed; this results in either the formation of mucous tissue, giving rise to a chondromyxoma, or the intercellular substance undergoes complete liquefaction and the cells are destroyed, in which case cysts are formed. In some cases the cartilage undergoes calcification, or genuine bone may be formed. In such cases we designate the tumor as osteochondroma. Excessive proliferation of the cells of the cartilage may result in the formation of sarcomatous tissue; then the neoplasm becomes a chondrosarcoma.

In the microphotograph presented we note all sizes and forms of cartilage cells, which are arranged in groups, each of which is surrounded by a capsule. Around several groups is another capsule continuous with the capsule which extends around the entire cartilaginous mass. Fat drops are seen here and there. The subepithelial tissue shows a marked infiltration with round cells (2). At the lower limit of the picture may be seen the epithelial covering (3) of the tumor.

V.

METASTATIC CARCINOMA OF THYROID IN NOSE.

There is little necessary, here, in the way of description; the picture speaks for itself. We see innumerable carcinoma cells arranged in groups or plugs, surrounded by a connective tissue stroma.

VI.

In this picture we have a higher magnification of No. V.

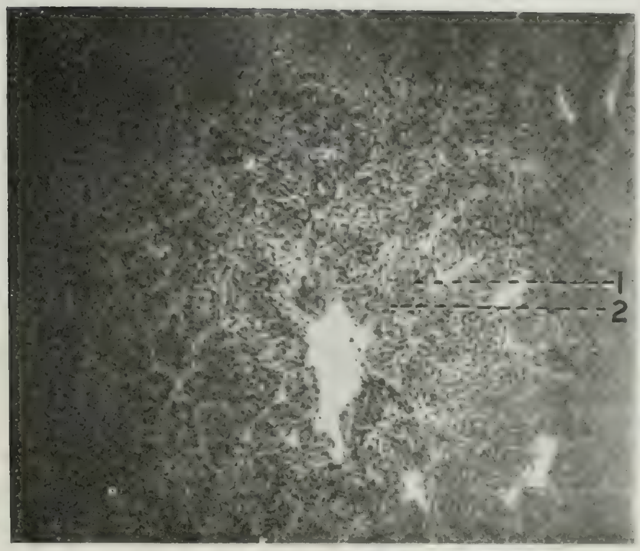

FIGURE 5.

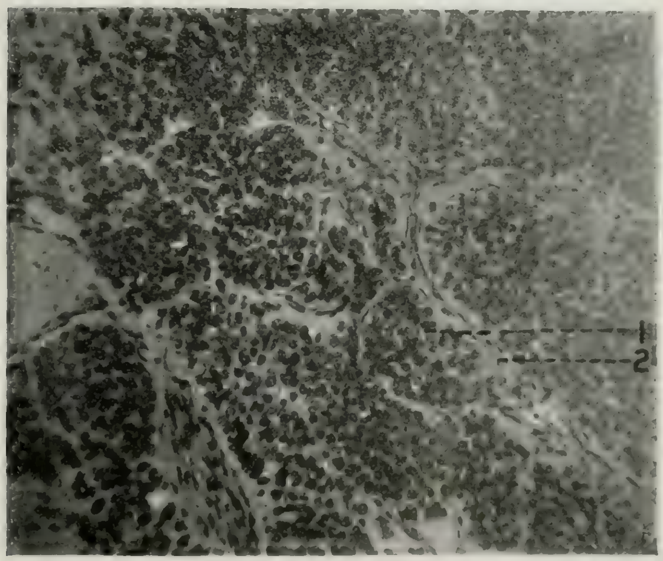

FIGURE 6.

VII.

MUCOUS CARCINOMA OF NOSE.

Mucoid cancer or gelatinoid cancer owes its peculiar characteristics to the fact that the epithelial cells produce a mucus-like substance. The mucus production may begin during the earliest development of the growth. The material collects in the center of the cell nests, like the secretion of a gland. The cells which surround the mucoid material are later broken through, with the result that the cells are loosened from their support and crowded together. Ultimately the epithelial cells are entirely destroyed, and we have a condition resembling the cylindromata, a condition which may be classified with the corresponding sarcomata. One may speak of mucous carcinoma at this stage as a carcinoma cylindromatosum. It is probably better, however, to retain the original nomenclature of mucous carcinoma.

In the microphotograph of a section from a mucous carcinoma of the nose, we are first interested in the large white area (1) on the right, which shows an advanced transformation of carcinomatous tissue into mucoid substance. Remains of epithelial cells are still seen suspended in the mucoid material. On the left may be seen an area (3) in which an earlier stage of the process is depicted. Here can be seen how the carcinoma cells are being separated by the collection of mucus. In other parts of the picture are to be seen nests of cells (6) which have not yet been affected by the collection of mucoid material. On the left is a large projecting carcinomatous mass (4) showing ulceration. Two varieties of epithelium are seen in the picture: above, pseudostratified ciliated epithelium (2); below, stratified squamous epithelium (5).

VIII.

This picture is an enlargement of the projecting carcinomatous mass in No. VII. At (1) are numerous carcinoma cells with practically no mucoid material around them. In the center of the picture is seen a large collection of mucus (2) which has separated the cells in that area; practically the entire projecting mass shows ulceration.

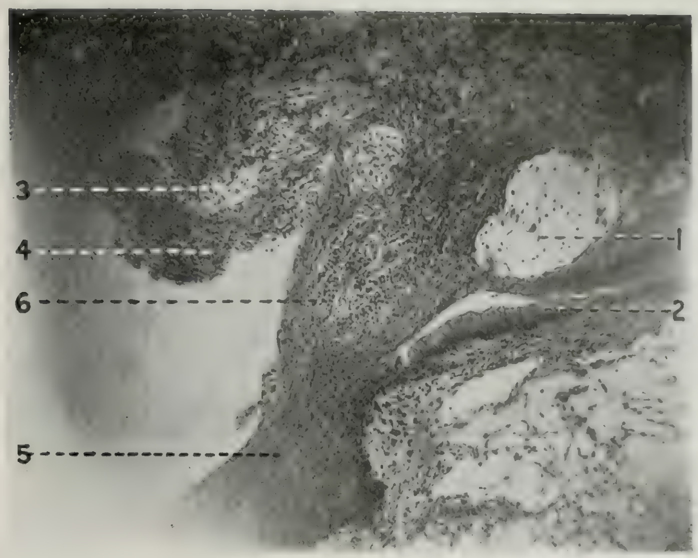

FIGURE 7.

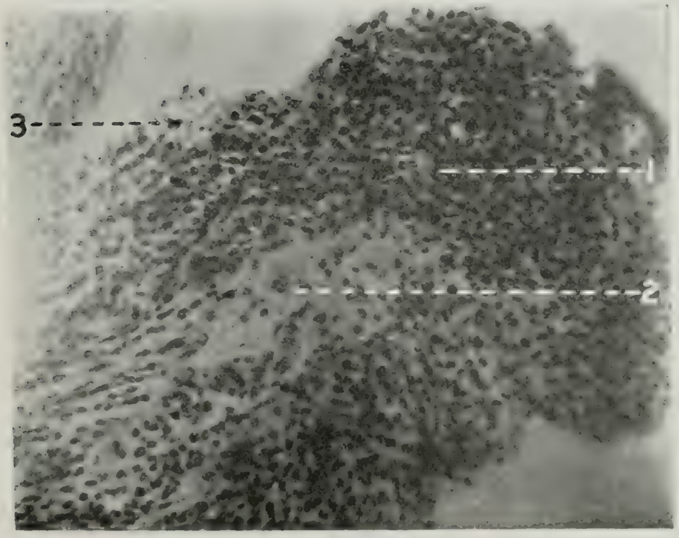

FIGURE 8.

IX.

MELANOSARCOMA OF NOSE.

A sarcoma is a connective tissue tumor in which the cellular elements are much more prominent than the intercellular substance. The sarcomata are closely related to undeveloped connective tissue. Sarcomata develop either in previously normal tissue, or in a connective tissue tumor. The transformation of the original tissue into the tissue of the neoplasm takes place by growth and multiplication of the existing cells. To consider the numerous types of sarcomata even briefly would result in a monograph beyond the object of this paper. A brief description of the type of sarcoma presented must suffice.

Melanosarcomata are developed in tissues which contain pigmented connective tissue cells—chromatophores. Metastases are common.

The microphotograph presented is of a specimen of metastatic melanosarcoma of the nose. Throughout the picture can be seen the pigment (2) which in the specimen is yellowish brown in color. The sarcoma cells are chiefly of the small round variety. There is some evidence in the right upper part of the picture of alveolar formation. In the remainder of the picture the sarcoma cells (1) show no regular arrangement.

X.

In this picture we have another field from the same specimen. Pigment is here much less in evidence. On the right is an area of ulceration (3). The alveolar arrangement of the sarcoma cells (1) is more manifest than in No. IX. In this field are seen, with a few exceptions, only small round cells.

In the lower portion of the microphotograph may be seen stratified squamous epithelium (2).

XI.

DERMOID CYST OF MOUTH.

The occurrence of tissue formations in localities where such formations are not normally met with can be explained by the assumption of embryonic misplacement of cells.

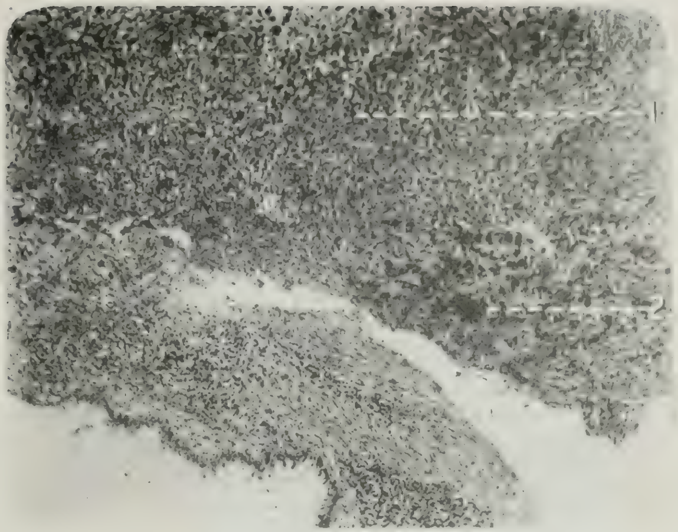

FIGURE 9.

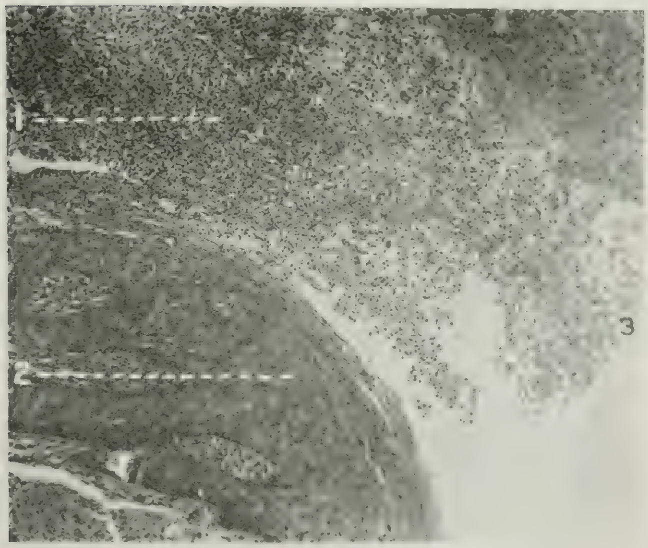

FIGURE 10.

Teratoid cysts may be divided into two chief groups: the ectodermal epithelial cysts on the one hand, and the entodermal and mesodermal epithelial cysts on the other. The ectodermal cysts vary in size from a centimeter in circumference to the size of a large fist. Their walls show ectodermal characteristics. The sac may consist of connective tissue lined with stratified flat epithelium. Such cysts are called epidermoids. The sac in other cases may present the characteristics of true skin: that is, it may show papillæ like those of the skin, with sebaceous glands, hair follicles, hair, sweat glands, and sometimes subcutaneous fat. These formations are called dermoid cysts.

The contents of such cysts are composed of either desquamated squamous epithelial cells alone or with fat and hair.

In the microphotograph presented we note superiorly the cavity (1) of the cyst. On the right, in the cyst cavity, is a mass of desquamated epithelium (2). Most of the contents of the cyst were lost during the preparation of the specimen. The sac is lined with stratified squamous epithelium (3) showing hornification. Three large papillæ extend toward the cavity of the cyst. In the particular field presented the sac shows little more than a dense connective tissue formation beneath the epidermal lining.

XII.

ANGIOFIBROMA OF GUMS.

We designate as angiomata those new growths in the structure of which blood vessels or lymph vessels constitute such an important part as to determine the character of the tumor.

A fibroma is a growth composed of connective tissue.

In the microphotograph presented we see a connective tissue new growth containing many newly formed blood vessels: hence the name angiofibroma. The most prominent element in the specimen is the mass of dense connective tissue (3) showing a moderate leucocytic infiltration (1). Throughout the picture may be seen numerous blood vessels (2) of different size. These vessels, with a few exceptions, are surrounded by a dense wall of fibrous tissue which is darker than the surrounding connective tissue.

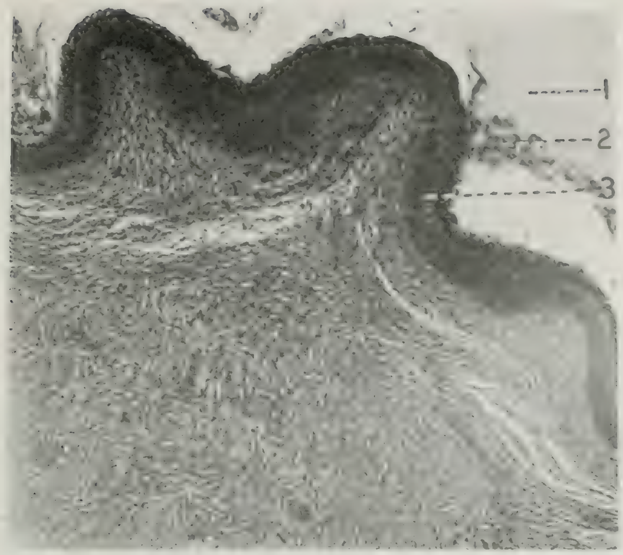

FIGURE 11.

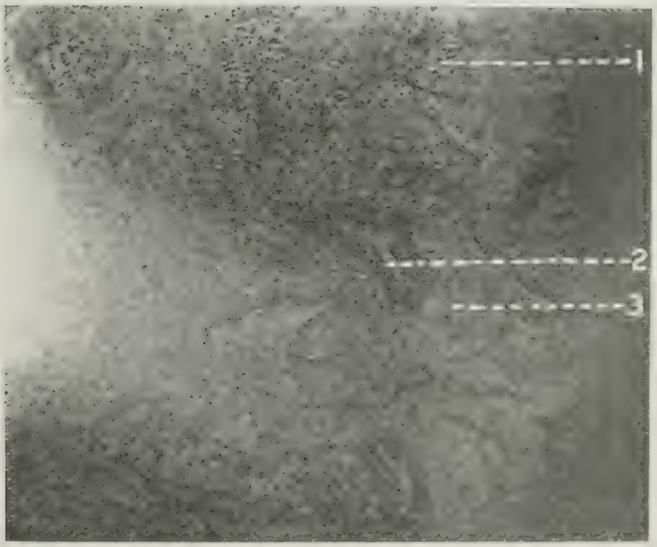

FIGURE 12.

XIII.

DIPHTHERIA PHARYNX.

In diphtheritic inflammation the greatest sufferers are the epithelial cells. A direct division of the nuclei occurs first; following this, necrosis. Finally, the epithelial cells become part of the false membrane which is formed on the surface. Marked leucocytic infiltration occurs, both in the subepithelial tissues and in the epithelial layer itself. The gland epithelium shows the same changes. Complete disintegration of some of the glands is at times observed. Exudation from the blood vessels in the subepithelial tissues results in an infiltration of the epithelial layers with a separation, in places, of the epithelium from the underlying supporting tissue. This separation results in the formation of vesicles. The deposit of fibrin results in the formation of the false membrane and in the subepithelial tissues, in the filling and compression of lymph spaces. In addition to fibrin the false membrane is composed chiefly of leucocytes, red blood corpuscles, and cellular débris. The blood vessels and lymph spaces are dilated. Muscle fibers become swollen and indistinct, and frequently show degenerative changes. Ulceration does not occur, as a rule, except in the larynx, as a result of prolonged intubation. After the separation of the membrane, repair takes place rapidly through the growth of epithelium from sound epithelial rests. Subepithelial regeneration occurs after the products of degenerative changes have been absorbed.

The microphotograph presented was taken from a specimen of diphtheria of the pharynx. Superiorly is to be seen the false membrane (1) infiltrated with leucocytes, red cells, etc. Several areas in which separation of the membrane (5) is occurring are to be seen. Immediately underlying the false membrane is an intense leucocytic infiltration (4), which is seen to extend directly through into the exudate above. In places remains of epithelium (2) are present. Necrosis of epithelium (3) can be seen in many areas. Beneath the muscle fibers (7) which are infiltrated with leucocytes, etc., and indistinct from degenerative changes, one notes many markedly engorged blood vessels (6). It is very interesting to examine these blood vessels closely for the exudation which was occurring at the time of death. In the lower portion of the picture one sees evidence of advanced degenerative changes in the glands (8).

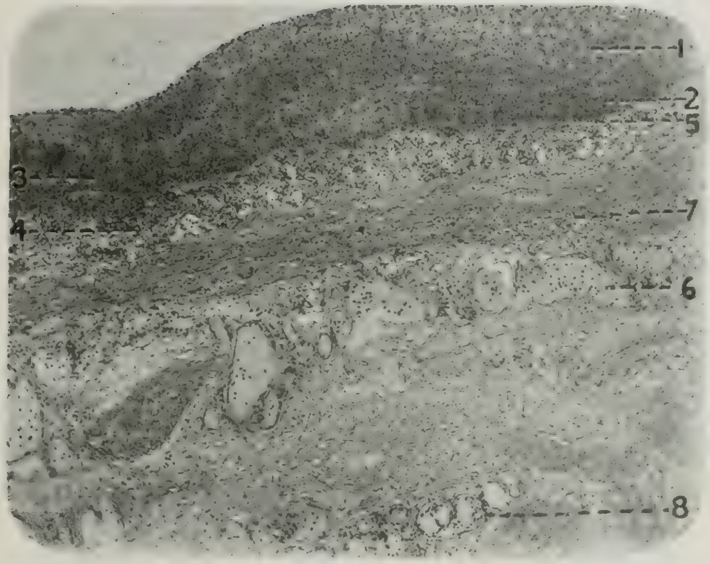

FIGURE 13.

XIV.

LYMPHOSARCOMA OF TONSIL.

Sarcomata and epitheliomata are both met with in the pharynx. The commonest form of sarcoma in this region is the lymphosarcoma, or small round cell sarcoma which frequently has its origin in the tonsils. Less frequently the sarcomatous cells are spindle shaped.

The lymphosarcomata imitate in structure the lymphatic glands. The stroma which holds together large numbers of round cells is composed of a vascular reticulum, a part of which is made up of branching and anastomosing cells. Depending upon the development of the reticulum, the lymphosarcomata may be divided into two forms: the soft and the hard. · In the hard variety the reticular framework may present a close resemblance to ordinary connective tissue.

In the microphotograph one sees on the right a lymph follicle (1) resembling very closely a normal tonsillar follicle. Upon closer observation, however, changes indicating the new growth will be noted.

These changes are chiefly in the grouping of the cells and the presence of a lymphosarcomatous reticulum. On the left in the picture is a mass of sarcomatous cells, both of the small round and the spindle shaped varieties.

XV.

CHRONIC LARYNGITIS.

The most important changes in the larynx resulting from a chronic inflammatory process are metaplasia and hyperplasia of the epithelium, increase in the fibrous elements, and subepithelial cellular infiltration. The epithelial hyperplasia is often very marked, especially in the posterior commissure: such a condition is known as pachydermia.

In the accompanying microphotograph one notes not only a metaplasia of epithelium into stratified flat cells, but a marked hyperplasia with hornification upon the surface. In the subepithelial tissues there is a marked fibrous tissue hyperplasia and a widespread round cell infiltration.

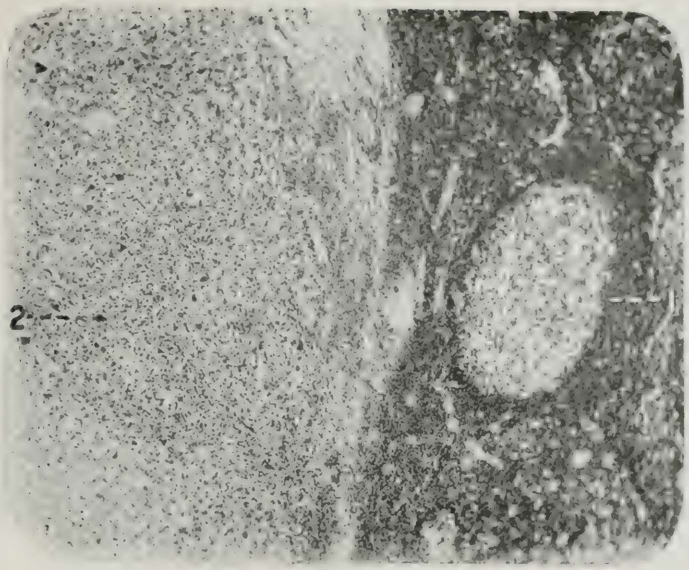

FIGURE 14.

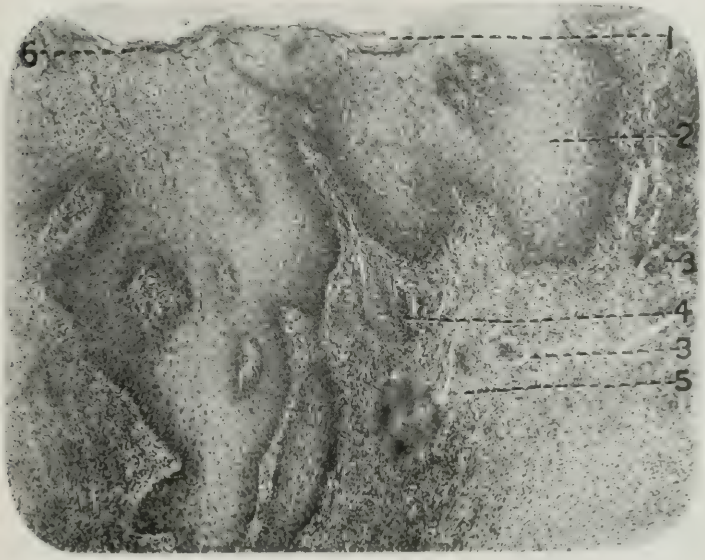

FIGURE 15.

XVI.

CYLINDROMA OF LARYNX.

As a detailed account of the formation of cylindromata has already been given in this paper in connection with another specimen, the writer will at this place simply describe the accompanying microphotograph. Three large cylindromatous spaces (1), filled with a hyaline mass (2), with some cellular elements scattered about, are seen with several smaller spaces above and below. Throughout the specimen are seen large masses of fibrous tissue (3) separating groups of sarcoma cells (4) and extending around the cylindromatous areas.

XVII.

LYMPHATIC LEUKEMIA LARYNX.

In lymphatic leukemia we may have in the larynx either a circumscribed or a diffuse lymphocytic infiltration. In the first picture of this condition we see on the left more or less circumscribed subepithelial infiltration (4). The stratified columnar ciliated epithelium (1) shows no change in structure and no infiltration. In the lower portion of the picture are mucous glands (2). Between the glands will be noted an intense lymphocytic infiltration (3). On the right is an area of edema (5) practically free of cellular infiltration.

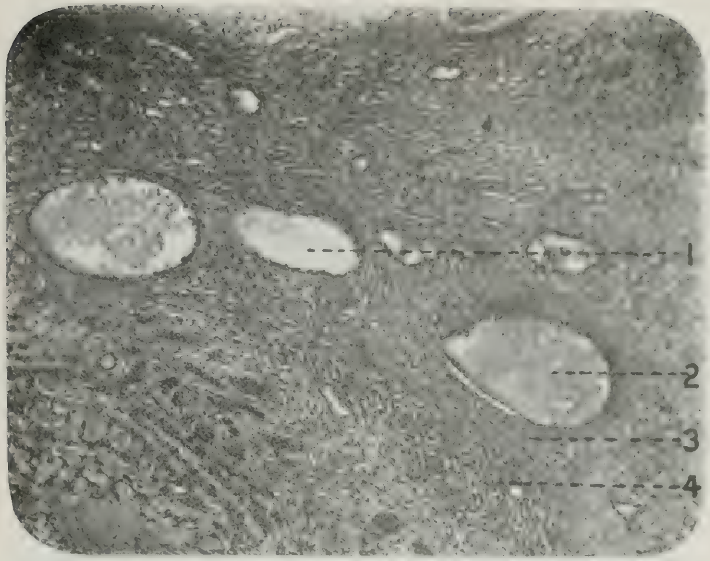

FIGURE 16.

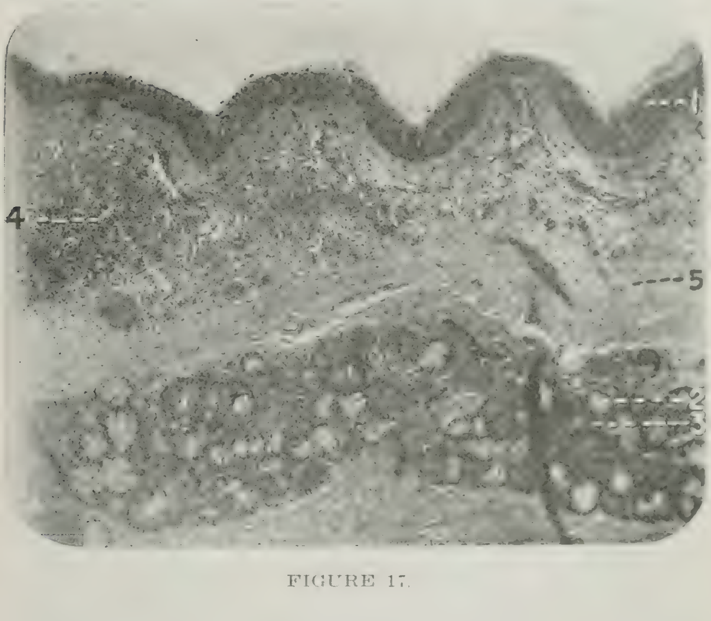

FIGURE 17.

XVIII.

LYMPHATIC LEUKEMIA LARYNX.

In this microphotograph one notes a diffuse subepithelial lymphocytic infiltration (2) and a metaplasia of epithelium (1).

XIX.

LYMPHATIC LEUKEMIA LARYNX.

Again in this picture one sees a diffuse subepithelial lymphocytic infiltration (3). Although the infiltration does not extend into the epithelium, excepting a few scattered cells, one notes in certain of the papillæ (1) as intense an infiltration as in the deeper tissues. Metaplasia of epithelium (4) is again noticed in this picture. Throughout the specimen are newly formed blood vessels (5).

XX.

CYST OF CORD.

Cysts in the larynx region are seen on the anterior surface of the epiglottis, on the cords, or on the lateral walls. Glandular cysts are rare. They result from a dilatation of gland acini

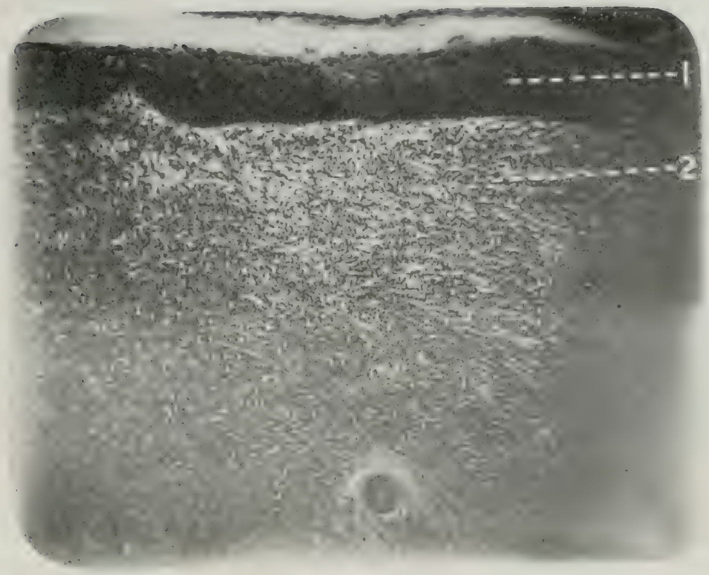

FIGURE 18.

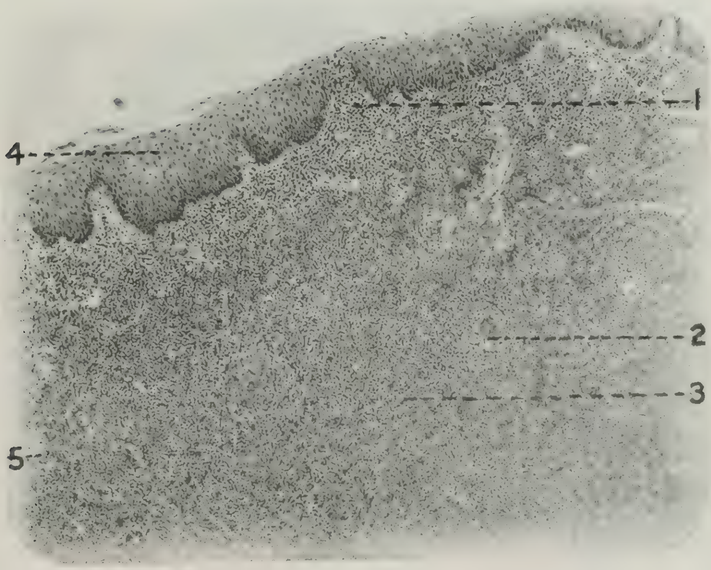

FIGURE 19.

or ducts, and are lined with glandular epithelium. After prolonged pressure the epithelial lining may be so altered as to be hardly recognized, or the original lining may be replaced by fibrous tissue. Another form of cyst occurring in this region is the connective tissue cyst. This may result either from a localized effusion of serum between the epithelium and underlying tissues, or to an effusion of serum into the subepithelial structures. Such cysts contain fibrous bands, hence the name.

The microphotograph presented shows a glandular cyst of the cord. The lining epithelium (3) has been flattened by prolonged pressure. The cavity of the cyst (1) is below and to the right. Remains of the cystic content (2) may be seen in the lower right hand corner of the picture.

XXI.

DIFFUSE CONGENITAL PAPILLOMA OF LARYNX.

Papillomata are primarily the result of epithelial hyperplasia. They consist of epithelial projections growing from a base of epithelial proliferation which has also resulted in the sending out of prolongations into the subepithelial tissues. The type of cell on the surface is usually that of the pavement variety. When springing from a part of the mucosa covered with columnar epithelium the cells, especially below the surface of the papillomatous projection, may resemble the type of epithelium from which they have grown. Each projection consists of layers of epithelium, with blood vessels and connective tissue in the center.

In the microphotograph one observes laryngeal tissue in the lower portion of picture. Upon the surface of this tissue are both stratified columnar (4) and flat epithelium. Subepithelially are glands (5). In the upper portion of the picture is the papillomatous tissue in which may be seen blood vessels (1), connective tissue (2), and the epithelium (6) of the papillomatous projections. Cross sections of the projections are seen on the right. Unfortunately the picture does not show the papillomatous projections springing from the laryngeal tissue.

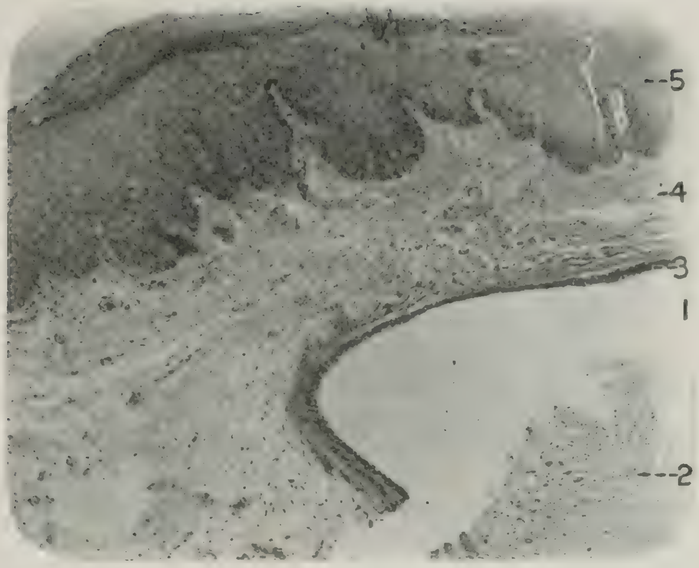

FIGURE 20.

FIGURE 21.

VI.

THE TONSILS AS AN ATRIUM OF INFECTION IN POLIOMYELITIS.

By Ernest M. Seydell, M. D.,

Wichita.

The recent reports of Mathers, Nuzum, and Rosenow on the etiology of anterior poliomyelitis which seem to show definitely that the organism is not a filterable ultramicroscopic virus, as previously considered by Landsteiner and Lavaditi, and Flexner and Lewis, but very probably a micrococcus, have opened some new avenues for investigation. Since it has been shown that in a number of cases the same organism found in the spinal fluid and tissues of the brain and cord has also been found in the tonsils of these same cases, it naturally makes one ask the question: Are these the portals of infection? Anterior poliomyelitis is an infectious disease accompanied in a certain proportion of cases by paralysis. The percentage of cases in which paralysis occurs is a great deal lower than has been previously believed—perhaps one in four.

BACTERIOLOGY.

In 1905, Geirsvold,[1] in a preliminary communication, stated that he had successfully isolated from the cerebrospinal fluid in twelve acute cases of poliomyelitis a Gram positive coccus, occurring in pairs, short chains and tetrads. He stated that he was able to make cultures of this microorganism on a variety of mediums; and further, he was able to produce definite paralysis in white mice, rabbits and pigeons. Landsteiner and Poppen,[2] and, working independently, Flexner[3] and Lewis determined that the virus of poliomyelitis passes through a Berkfeld filter, and were able to produce a disease in the monkey which, in its clinical, pathologic and histologic features, was quite similar to acute poliomyelitis in man. They were unable to produce the disease in mice, rats, guinea pigs, dogs, cats, goats, sheep or horses. In 1913, Flexner and Noguchi[4] cultivated and demonstrated, microscopically, a small filterable microorganism with which they produced poliomyelitis in monkeys. The cultural conditions were similar to the bacteria, but they did not classify the microorganism—rather inclining to the view that the disease was caused by a virus. In September of this year Mathers[5] published a report in which he stated that he had isolated a Gram positive micrococcus from the brain and cord of fatal cases occurring in the Chicago epidemic. He obtained these organisms in six out of seven cases examined. He did not obtain the micrococcus from the spinal fluid or heart blood. In closing, he stated that in view of the accepted fact in regard to the filterable virus of epidemic poliomyelitis, it would seem most reasonable to regard the micrococcus as a secondary invader. A short time after this Nuzum[6] and Rosenow,[7] working independently, Nuzum in Chicago, Rosenow in New York, published the results of their observations, both having undoubtedly discovered the same organism. Nuzum found the organism in each of the twelve cases examined by him. The micrococcus was obtained from the brain and cord at necropsy. He also reported the finding of this same micrococcus in the spinal fluid during life in eight out of nine cases examined. Since then these examinations have been extended, and in a subsequent report,[8] he adds that he has found the same organism in forty-five out of fifty cases, or in ninety per cent. Twice he cultivated the micrococcus from the heart blood of the patient during life. The organism has been found in the spinal fluid twenty-four hours after the onset of the disease, and may still be found there two months after the patient has apparently recovered. Rosenow first, and later Nuzum, demonstrated these organisms in the tonsils of patients suffering with poliomyelitis. The former also found them in the ventricular fluid after death, from the blood during life in one instance, but not from the spinal fluid. He also isolated the organism from the brain and cord in each of twelve cases examined. The micrococcus is polymorphous —it appears in a minute form on anaerobic cultivation, and readily grows out into a larger form when cultivated aerobically.

They usually are found in short chains and clumps, and also in diplococcus arrangement. The smaller forms can be passed through a Berkfeld filter, and the subsequent cultures show both the large and small forms of the organism.

ANIMAL EXPERIMENTS.

In 1913, Flexner and Noguchi produced poliomyelitis in monkeys with their filterable virus. Rosenow produced paralysis with lesions in the central nervous system in guinea pigs, rabbits, dogs, cats and monkeys by intravenous and intracerebral injections. He produced the same lesions by injecting emulsions of pus expressed from the tonsils, also emulsions of the extirpated tonsils and brain tissues. Nuzum made cultures from the tonsil crypts of eleven cases of acute poliomyelitis and from eight pairs of tonsils removed from patients suffering with the same disease;[9] and from all of the above, with the exception of two cases, the same peculiar Gram positive microorganism was isolated repeatedly, not in pure culture as a rule, however. With these cultures he produced paralysis, etc., in fourteen out of seventeen rabbits inoculated. In one Macacus Rhesus monkey and one small ring tail monkey injected with the tonsil cultures, there have been no results to date. Positive results were obtained in rabbits with cultures from the nasal discharge of two cases, from emulsions of brain tissue in two others, and also in one case using mesenteric lymph gland emulsions. The monkeys developed the typical lesions when injected with cultures obtained from the spinal fluid. The rule that the younger animals are more susceptible to inoculation holds true as it does in the human race. It may be said that the incubation period varies from thirty hours to fourteen days, also that the picture produced in the monkey is typical of acute poliomyelitis, both clinically and pathologically. In young rabbits there is a variation in the microscopic picture, although many of the changes attendant on the disease in man and monkeys are present in the central nervous system after paralysis.

THE ATRIUM OF THE INFECTION.

Now arises the question, where do these germs enter the system? I might say that it is impossible to definitely answer this most important query at this time, but I believe, from the evidence already accumulated, that the tonsils play an important rôle in this direction. It has been the supposition for a number of years that the infection is disseminated from the nasopharynx of the diseased to the nasopharynx of the unfortunate victim. In America, Sohier Bryant,[10] Stephens,[11] Anderson and Frost, as quoted by Bryant, Sheffield,[12] and others state that in many cases acute poliomyelitis is preceded or accompanied by nasopharyngeal inflammation, and suggest that many cases presenting a nasopharyngitis during an epidemic may be an abortive type of this disease. Experimental work should now be undertaken to prove the truth of these assertions. Rosenow[13] states that the tonsils in patients suffering with poliomyelitis, especially those over three or four years of age, yield on pressure a surprisingly large amount of infectious material even where no objective symptoms of tonsillitis are present. These tonsils present numerous areas of localized necrosis, usually along the capsule and not communicating with the surface. They contain a peculiar opalescent material in which are found large numbers of the micrococci. These have also been found in the adenoids. In approximately twenty-five cases of infantile paralysis in which the temperature remained high and the paralysis was progressing, tonsillectomy was performed. No deleterious effects were observed in any of these cases; on the contrary, it was found that following tonsillectomy the temperature abated and convalescence was rapid. The results were so striking in some of the above cases that Dr. Rosenow suggests the advisability of removing the tonsils in this type of cases. The next question which naturally arises at this place is: How many of these cases that have had poliomyelitis have previously had their tonsils removed? To help determine this important question Dr. Meyer and the writer have carefully analyzed the histories of over two hundred cases which were treated at the Cook County Hospital during the recent epidemic. Our findings may be tabulated as follows:

Total number of cases studied, two hundred and three.

Average age, four years.

Oldest patient suffering with the disease was fifty years of age.

Youngest patient was six weeks old.

Deaths in this series numbered ten, or five per cent.

Average age of fatal cases was four and one-half years.

Oldest patient to die was twenty-six years.

Youngest, ten months.

In this number twenty-one cases, or ten per cent, had some cervical adenopathy.

Fifteen cases, or seven and one-half per cent, had an angina as an initial symptom.

Ten cases, or five per cent, had some discharge from the nose.

I might state that the Chicago Board of Health figured the mortality at twenty-one per cent. This was reduced in this series by the fact that some of the cases diagnosed as poliomyelitis proved to be tubercular meningitis or brain tumor at postmortem. In the two hundred and three cases whose histories we examined, we find that in the majority of the cases the tonsils were recorded as small. In a very much smaller number they were stated to be enlarged and slightly congested. In two cases one tonsil had been removed, and in one other a tonsillectomy had supposedly been performed. Upon examination, however, I found some tonsil tissue on the right side which contained a large crypt, and also found an adenoid. In these cases where a portion of the tonsil tissue had been removed the paralysis was slight and the convalescence rapid. In a communication from Dr. Rosenow on this subject he states: "I know of two cases that have developed poliomyelitis in whom tonsils and adenoids were presumably properly removed. In one, there was only a slight weakness of the shoulder muscles, which disappeared promptly. In the other case the paralysis was slight, even though a brother of the patient, whose tonsils had not been removed, was severely paralyzed. I have seen these two cases, and have heard of a few others, and I believe in all cases the paralysis was not marked."

CONCLUSION.

A review of the findings of the Chicago epidemic do not lead one to believe that many of the cases had a nasopharyngitis, neither as an initial symptom or later in the disease. It must be remembered, however, that many of these cases were brought into the hospital with the disease well advanced, and it was sometimes very difficult to obtain any definite history. In this epidemic the organism did not produce cervical adenopathy; neither did it seem to produce apparent inflammation of the mucous membrane covering the tonsils, as in most instances in which abscesses were found the tonsils appeared normal.

Three investigators, working independently in widely separated cities, have recently isolated what appears to be the same peculiar microorganism from the tonsils, tonsil crypts, spinal fluid, brain and cord, and even from the heart blood and mesenteric lymph glands of cases of poliomyelitis. Their experimental reports indicate that the micrococcus has met all of the provisions of Koch's laws.

It has been known for a long time that the causative factors of poliomyelitis remain in the nasopharynx of the patient for as long as six months. Now we find that we can produce the disease in animals by injecting them with the contents of abscesses found in the tonsils of the affected patient. The disease attacks young children in a very high percentage of the cases, who, as we well know, have a tendency towards abnormal conditions of their lymphatic systems. One of the striking features of this disease is the elective affinities of this organism for the nervous system on the one hand, and for the lymphatic system on the other. The germs seem to travel to the central nervous system along the perineural lymph channels.

In the two hundred and three cases which we studied we find tonsils present in two hundred cases. In three cases there was a partial absence of these structures, and in these cases the paralyses were slight and recovery rapid. In the cases of Rosenow we find a similar course and result.

All of which very greatly strengthens our belief that the tonsils and adenoids are the portals of infection of this alarming disease.

In closing, I wish to thank Dr. Nuzum and Dr. Meyer, who made it possible for me to publish the reports of the Cook County Hospital.

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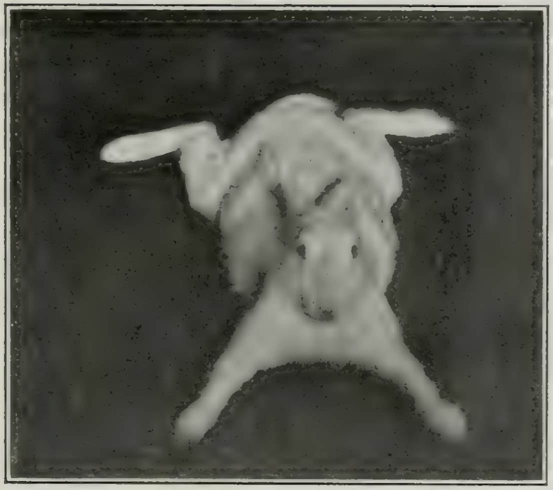

FIGURE 1.

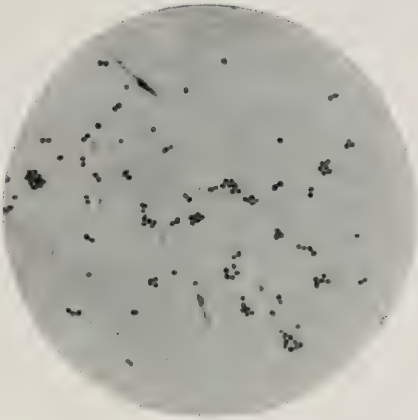

FIGURE 2.

Twenty-four-hour culture from the spinal fluid. Small variety.

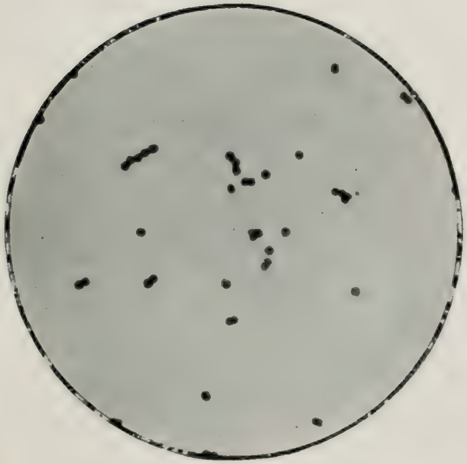

FIGURE 3.

Seventy-two-hour culture from the tonsil. Large variety.

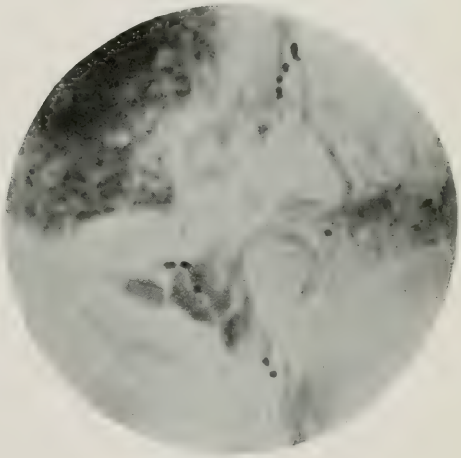

FIGURE 4.

Micrococci in nerve tissue.

VII.

TUBERCULAR MASTOIDITIS—RADICAL OPERATION UNDER COCAIN ANESTHESIA.*

By Harold Hays, M. D.,

New York.

About two years ago, Mr. S., aged twenty-four years, came to consult me about his deafness and sharp pain in the right ear. His history showed that he had had a tuberculosis of the lungs which at that time was in a quiescent state. He had had a radical mastoid operation performed on the left ear and mastoid four years before. This had never healed properly, with the result that there was a large opening into the dura which was constantly suppurating.

Examination of the right ear showed granulations in the middle ear, entire destruction of drum, and the discharge of cheesy-like pus. Culture from this ear and from the sinus behind the left ear showed numerous tubercle bacilli. He could hear neither watch nor tuning fork at either ear.

During that first winter I had him under observation two or three times a week, during which time I attempted every known means of clearing up the condition.

As he did not improve, I advised a radical mastoid operation.

The question of anesthesia was an important one. Here was a patient with a quiescent tuberculous process in his lungs, a patient whose general physical condition was far below par. Ether anesthesia per oram would possibly light up his lung condition, so I determined to operate upon him under local anesthesia.

On the day of the operation at ten o'clock in the morning he was given a hypodermic injection of morphin grain one-fourth, atropin grain one-one-hundredth. Two hours later a second injection of like amount was given. At two o'clock he was brought to the operating room. As there were no signs of opium poisoning, another injection of morphin grain one-eighth was given. The operative field was prepared in the usual manner. A solution of equal parts of ten per cent cocain with equal parts of adrenalin having been prepared, an injection of this solution along the line of the proposed incision was made, going well into the surrounding tissues, forward to the lobe of the ear, upward to the zygoma, and backward to the occipital bone. An attempt was made to have the injections reach down to the periosteum. This is not a difficult matter in this region, as the tissues are not very thick.

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*Read before the New York Academy of Medicine, Section on Otology, October 13, 1916.

The semilunar incision was made throughout its whole extent down to the periosteum. A longer needle was attached to the hypodermic syringe and an injection made under the periosteum of a one-half of one per cent cocain solution with equal parts of adrenalin (Shleich infiltration). Two to three drams of this solution were injected, care being taken that some of it reached the periosteum below the tip and anterior to the bony canal. It was spread through the tissues posteriorly and above until an area of at least three to four square inches was infiltrated.

The periosteum was then incised and scraped back with a rasp and the retractors were inserted.

After the bone was well exposed the mastoid cavity was uncapped with a chisel. The patient complained of no pain at any time while I was working in the mastoid cavity. The operation was a Schwartze-Stacke. The cavity was extremely large, the bone extremely hard, the tip entirely destroyed so that it had to be removed in its entirety. When the patient was asked to describe his sensations, he stated that he felt no pain, only the apprehension of pain, similar to what he would feel in the dentist's chair. We are wont to feel that in the mastoid operation under ether anesthesia, the constant chiseling and using of rongeurs produces a slight concussion of the brain, which accounts for the lessened amount of ether we have to give. After this experience I am inclined to think that this idea of ours is visionary. The constant pounding of the bone produced no marked change in the patient's mastoid condition. In fact, he did not seem to mind it at all.

The mastoid cavity was thoroughly cleaned out without any trouble. The bony canal wall and the bridge over the antrum were taken down without any pain. However, when the sensitive mucous membrane of the middle ear was reached, trouble commenced. It had been impossible thus far to cocainize this membrane. Here was a mass of sensitive granulation tissue. In order to overcome this sensitiveness a four per cent cocain solution was injected along the posterior canal wall, into the granulations themselves and deep down into the eustachian tube. Not all sensitiveness could be overcome, but enough to proceed with the operation with little trouble. The mass of granulations were removed with a curette until I came to a thicker tougher mass, apparently attached to the internal wall of the middle ear. I hesitated to remove this, as I realized that the facial nerve was either in it or close by. But the mass was infected and had to be removed. I grasped it with forceps and snipped it off with a small scissors. At once the patient exclaimed: "Doctor, you must have hit my facial nerve. The right side of my face is paralyzed." Such was the fact.

The eustachian tube was curetted and a skin flap for a new canal made without any trouble. The making of the skin flap was facilitated by the lack of bleeding, due to the blocking off of the vessels by the cocain solution. When one realizes how much bleeding there usually is, he can appreciate of how much value this ischemia was. After making the artificial canal the posterior wound was closed with tendon sutures.

Three hours after the operation the patient sat up in bed to eat his supper. Having had a radical mastoid operation performed on the other side under ether anesthesia, he was in a position to make comparisons. He was most enthusiastically in favor of local anesthesia. Not only had the general reaction been severe at the time of the former operation, but his wound pained him a great deal. In this instance the pain was practically nil. At the end of the week he left the hospital with his wound in excellent condition. The facial paralysis began to improve, so apparently the nerve was not completely severed.

A few months ago I lost track of the patient. I had hoped to present him at this meeting. But I received a letter a short time ago stating that he had died at Bellevue Hospital during the summer from a tubercular meningitis. Autopsy showed a direct extension of tuberculosis from the suppurating cavity behind the left ear to the dura.

The practical points to be deduced from this case are the following:

1. That the radical mastoid operation can be done under local anesthesia without any pain.
2. That the superficial scalp tissues and periosteum are sensitive, but that bone has absolutely no sensation.
3. That the mucosa of the middle ear is extremely sensitive and must be separately cocainized.
4. That irritation or destruction of the facial nerve is immediately noticeable to the patient.
5. That the after-affects are practically nil.
6. That the end result is just as good under local as general anesthesia.

VIII.

SALIVARY FISTULA FOLLOWING A SIMPLE MASTOIDECTOMY WITH CERVICAL ABSCESS.

By Frederick C. Schreiber, M. D.,

Washington, D. C.

Of the very many postoperative sequelæ following a simple mastoidectomy, that of a salivary fistula is one of the most rare, and paralysis of the facial nerve a much more frequent complication. It is quite natural, then, that a case incorporating these two conditions should be regarded as unique. More particularly is this true when it is understood that both entities were sustained by a common and remarkable injury to the lower lobe of the parotid gland. The case I am to present this evening entered the New York Eye and Ear Infirmary, on the service of Dr. Lewis, with the following history supplied by the mother:

L. S., female, aged seven years, with negative family history, was operated on five years ago for an acute mastoid abscess with cervical abscess. She had suffered with a cold for ten days prior to the onset of a double acute otitis. Three days later a large swelling appeared in the upper left cervical region. The child was taken to a dispensary, where immediate operation was advised. Two weeks after the operation the child was dismissed, and in three weeks more the mastoid had completely healed with the exception of the fistula still existent. Unfortunately, these facts could not be corroborated by the hospital to which credit for the operation was given.

Examination of the patient's operated ear discloses evidence of a straight incision, fairly well back from the auricle. The lower part of the incision is carried downwards and forwards to just below the angle of the jaw. The incision is completely healed with the exception of a minute point, which can be seen at the angle of the jaw periodically discharging a slightly turbid, colorless fluid. The fluid increases with mastication and deglutition, although it is not as dependent on the latter as the former. Articles of food not designed to excite salivary secretion are not accompanied by a profuse discharge from the fistula opening.

Examination of the fluid showed it to be a somewhat turbid, colorless and odorless secretion, with a tendency to viscidity. It was slightly alkalin to litmus, and showed nothing of importance microscopically. Psychic influences governed the rate of flow considerably. In a strange environment the flow was completely arrested, only to be reestablished by mechanical or chemical means. Of the methods used to promote a free flow, one above all the rest proved most satisfactory, that of chemical stimulation, employing citric and acetic acids, in the form of lemons and pickles.

The secretion was collected to determine its ability to split the polysaccharides into less complex sugars. With this in mind, a bit of starch was heated in a test tube of water to dissolve off the outer cellulose layer. Fehling's solution was used in the usual way to determine the presence of dissaccharides. A control was made, adding starch paste to the Fehling's, whereupon no reduction of the copper salt was observed. A small amount of the secretion gathered was placed in a test tube filled with about ten cubic centimeters of the paste. An equal quantity of the patient's saliva was added to the same amount of paste, and a comparison was attempted. It was found that the secretion collected exerted a much more positive hydrolytic cleavage on the polysaccharide than the saliva. In shortly less than eight minutes a definite reaction for sugar was detected by Fehling's reagent.

No reaction resulted when a solution of ferric chloride was added to the secretion, to determine the presence of the sulphocyacids which are supposed to be constantly present in parotid secretion.

The biuret test for mucin was next attempted and, strange to say, a positive reaction was recorded. This, in the light of the fact that the parotid gland is a true serous gland, supposedly not secerting mucin, seems a bit contradictory, and the author is at a loss to explain the reaction, unless the submaxillary gland is also involved.

The next test was undertaken to prove or disprove the communication of the fistulous tract with the pharynx. To this end it was decided to inject the tract with a solution of methylene blue or with bismuth paste, but the size of the punctum precluded such a possibility. Then the patient was instructed to gargle and later to swallow about four ounces of starch water. The fluid was collected for a period of fifteen minutes, but without success of a positive reaction to iodin.

The patient was then given six drops of a saturated solution of potassium iodid, with the idea of recovering some of the drug in the secretion. A solution of nitric and nitrous acids, to which an equal amount of starch paste was added, served as an indicator. The time of reaction was taken, and in twelve minutes a positive blue ring struck in the test tube, confirming the presence of the iodid in the fistulous fluid.

An attempt was made to probe the duct to determine its course. The finest Stenson's duct probe was employed, but it could only be passed to a distance of one-eighth to one-quarter of an inch, where its further progress was arrested.

Of the disease entities with which this condition could be confused, and from which it was necessary to differentiate, might be mentioned Potts' disease affecting the cervical vertebræ, with abscess formation discharging in the lateral cervical region, suppurative adenitis, and branchial fistula.

The thought of the first two disease entities, Pott's disease and suppurative adenitis, was dispersed by the result of the macroscopic, microscopic and chemical analyses of the secretion. However, the third condition seemed more probable: so plausible, in fact, that at first it was thought that the condition presenting was a branchial fistula.

To exclude the possibility of this, it became necessary to definitely prove that there was no communication with the pharynx. This would naturally presuppose that a blind external fistula had existed since birth, which had accidentally been opened into at the time of the operation, converting it into a complete pharyngocervical fistulous tract. The position of the opening, periodicity and nature of the discharge all argue in favor of a salivary fistula complicating a mastoidectomy. The diagnosis might even be further restricted to a parotid fistula affecting the parenchyma of the gland, with a remote possibility of a submaxillary involvement, which is substantiated by the physiologic characteristics of the secretion and the associated paralysis of the lower cervicofacial branches of the seventh cranial nerve, both occasioned by faulty technic.

In conclusion, I wish very superficially to review the importance of the parotid gland. This gland should engage the attention of every otologist and rhinolaryngologist from a physiologic, pathologic and anatomic standpoint. Physiologically it plays the premier rôle of the paired salivary gland in secreting the ptyalin so necessary to the clevage of the complex carbohydrates and in secreting a fluid very essential to moistening the bolus of food in order that it may stimulate the taste buds on the posterior part of the tongue. Pathologically this gland is of interest: First, because of the constitutional and reflex conditions which affect its secretory activity; second, because of the suppurative process, both circumscribed and phlegmonous, which may affect its integrity, and the relation it bears to Ludwig's angina; and third, because of the frequency of heterologous tumor formation within its substance, notably chondroma. It might be well to mention the clinical parallelism between acute circumscribed abscess formation pointing in the oropharynx to retropharyngeal abscess and abscesses pointing toward the external auditory canal to furuncular conditions, so prevalent in the cartilaginous portion of the meatus.

By far the most interesting and important feature of this gland is its anatomic position and the structure which it harbors. Extending, as it does, in front of the ear from the zygoma above to the lower border of the body of the mandible below, covering the posterior one-third of the masseter muscle, and extending backwards to the external auditory meatus, the mastoid process, sternomastoid muscle and the posterior belly of the digastric, it forms a most formidable structure constantly to be mindful of by the workers in this special branch of medicine. Add to this, then, the structures which pass through its substance, namely: (three nerves) facial, great auricular and auriculotemporal; (four veins) superficial, temporal, temporomaxillary with communicating branches to internal jugular; internal maxillary, postauricular, and one artery, external carotid, and its importance becomes even more manifest.

IX.

A CASE OF LABYRINTHINE FISTULA WITH COMPLETE LOSS OF COCHLEAR FUNCTION AND PERSISTENCE OF NORMAL VESTIBULAR FUNCTION.

By Julius Auerbach, M. D.,

New York.

This man is forty-three years of age, and gives a history of discharge from left ear since early childhood. For the past five or six years the ear has been practically dry, and aside from the impairment of hearing, he was in no way troubled until seven months ago, when he had a violent attack of dizziness accompanied with sensations of turning, nausea and vomiting, and disturbance of equilibrium; this lasted for a day, and was followed by another but less marked attack two weeks after the last attack.

Examination disclosed the following:

Right ear showed a normal drum membrane and normal hearing. In the left ear the drum membrane was entirely destroyed, the canal dry, and the inner wall of the tympanic cavity exposed and epidermized; the ossicles are gone; the eustachian tube is open.

Hearing tests with the Bárány noise apparatus showed total deafness to the loud conversation voice. Acoumeter not heard when held close to the ear. The Weber test showed lateralization to the nonaffected ear. The low middle and high forks not heard.

The Stenger test is negative, proving a complete unilateral deafness; the reading test, carried out with the noise apparatus, also shows complete deafness on that side.

Static Labyrinth.—Spontaneous nystagmus is absent.

(1) Turning Test.—Turning ten times to the right with closed glasses, in order to eliminate the error of optic nys-

tagmus, gives a nystagmus to the left lasting twenty seconds. Turning ten times to the left gives a nystagmus to the right lasting twenty-four seconds.

(2) Caloric Test.—In view of the otoscopic findings, this test with hot and cold water was not performed. I employed instead the Struycken apparatus with ethyl chlorid, and was able to elicit a nystagmus to the opposite side.

(3) Fistula or Mechanical Test.—Compression of air in the external auditory canal with a large olive closing the canal gives a typical nystagmus, with the quick component to the same side and a slow component to the opposite side. Aspiration reverses the nystagmus. The head motion is in the direction of the slow component.

I have said that the eustachian tube is open; when compression is carried out the patient feels a current of air in his throat; if the entrance of the eustachian tube into the tympanic cavity is closed by a pledget of cotton, much less compression is necessary to cause the same degree of nystagmus; or if the patient do a Valsalva at the same time that the test is carried out, a more violent nystagmus is elicited.

We have here then a case of complete unilateral deafness with preservation of the static labyrinth and the presence of a labyrinthine fistula, as is evidenced by the fistula test.

The cause of the complete deafness is not absolutely clear to me. Was it produced by a slow degenerative process of the cochlea nerve and the organ of Corti, or did the patient pass through a serous labyrinthitis at the time of his first violent attack of dizziness, with the consequent production of this total deafness?

Now as to the location of the fistula: Fistulæ of the semicircular canals have been found on the operating table where the previous fistula test proved negative. On the other hand, none has been found where the test proved positive.

The most favorable site for the formation of fistulæ is in the horizontal or external semicircular canal, this being the most exposed to suppuration in the tympanic cavity and antrum, it being remembered that this canal lies partly in the antrum.

In view of the fact that the ear has been absolutely dry, that there was no spontaneous nystagmus and no dizziness, I thought it advisable to keep him under observation. In the seven months that have elapsed he has attended to business, that of a shoe salesman, has not lost a day's work, and has been free from all symptoms.

The interesting features of this case are:

(1) The practically normal vestibular reaction, showing neither an increased nor a diminished static activity.

(2) Complete loss of cochlear function, indicating a widespread invasion of the labyrinth at some past period.

(3) Question of treatment. This patient was seen by several otologists, who advised a radical mastoid operation. Was this advice justified in the light of the findings?

(4) A dry ear with no spontaneous nystagmus, no dizziness, no loss of hearing seems to call for no operative interference.

(5) With a recurrent acute infection of the middle ear causing retention, dizziness, spontaneous nystagmus, and disturbance of equilibrium, a radical mastoid would be in order. In no case should a labyrinth operation be considered, unless intercranial complications were threatening or manifest.

THE READING TEST AS AN AID IN DIAGNOSIS OF COMPLETE UNILATERAL DEAFNESS.

This test is carried out with the aid of the Bárány noise apparatus as follows:

The patient is given something to read aloud in his usual conversational tone. With the noise apparatus inserted in the canal of the deaf ear, no impression is made on his reading voice; when, however, the apparatus is inserted in his good ear, his voice becomes louder and louder, compensating for the noise created by the apparatus, though the patient is unconscious of the fact that his voice is raised.

THE STENGER TEST TO DETERMINE COMPLETE UNILATERAL DEAFNESS, REAL AND SIMULATED.

This test is carried out with the aid of two equal tuning forks, preferably small A^1, and is dependent upon this phenomenon: that when both forks are sounded, the fork held close to the ear will completely obliterate the sound of the fork which is held farther from the ear. In complete deafness this phenomenon is reversed; that is, the fork, although held close to the deaf ear, is not heard, while the fork held at a distance from the good ear is heard. In simulated deafness the examiner stands behind the patient and sounds both forks at the same time; taking, for example, that deafness in the left ear is simulated, the patient will not hear the fork on the right side when one tuning fork is sounded and held close to his left ear, showing that he does perceive sound on the left side.

X.

RESULTS IN FOUR CASES OF A MODIFIED RADICAL OPERATION FOR CHRONIC PURULENT OTITIS MEDIA.

By Hugh B. Blackwell, M. D.,

New York.

The operative procedures performed in these four cases have been employed some nineteen times by me during the past three years. The purpose of the operations was to cause a cessation of the discharge, and at the same time to conserve or even improve the hearing.

The first patient, L. K., twenty-three years old, was admitted for operation to the Manhattan Eye, Ear and Throat Hospital six months ago, with a history of a discharging right ear for three years previous to admission. During all this time there was a more or less constant feeling of fullness or heaviness in the head. It was mainly to seek relief for this symptom that he came to the hospital.

Aural examination on admission revealed the presence of foul pus in the external auditory canal. Shrapnell's membrane was missing. By means of a large probe introduced through this opening, bare bone could be felt in the attic, and small portions of cholesteatoma were removed. The remainder of the drum membrane was intact. Weber was referred to the normal ear. With the Bárány noise apparatus in the normal ear, the patient was unable to hear the loudest shout in the affected ear. After injecting the affected ear with cold water for three minutes and forty seconds, a nystagmus reaction was produced. Rotation tests were normal.

Operation.—Modified radical. The cortex was very hard; there were practically no cells present in the mastoid, save in the antrum, which consisted of a single large cell or space filled with cholesteatoma and granulations. The posterior wall was taken down to the epitympanic ring; it was found to be necrotic and partially broken down, and was entirely removed. The short processes of the incus were missing. Granulations and cholesteatoma were removed from the internal and external attic without disturbing the ossicles, and the remainder of the drum was left in situ. A meatal flap was cut and sutured to the temporal fascia, and the posterior wound was sutured throughout.

Since operation the feeling of heaviness and fullness has disappeared, the ear is dry and has been so for the past four months. The patient can now hear an acoumeter in the affected ear at one foot, and a moderately low whisper at the same distance.

The interesting features of this case are: First, the marked deafness, apparently absolute, in the affected ear before the operation, associated with a normal vestibular reaction, and Weber referred to the normal ear; second, the return of hearing to the affected ear since operation; third, the cessation of the discharge.

Case 2.—M. A., nine years old, was admitted for operation to the Manhattan Eye, Ear and Throat Hospital six months ago, with the following history:

Seven years previously a mastoid operation was performed upon the affected ear; three years ago a second operation was performed on the same ear. The ear had been discharging ever since the first operation. A few days prior to admission to the hospital a swelling was noticed in the old cicatrix.

Aural examination on admission showed the presence of large quantities of foul pus in the canal. Posterior to the auricle there was a swollen red cicatrix, mainly in its inferior angle. On gentle pressure over the cicatrix pus was easily forced from the canal.

Operation.—Modified radical. On opening the mastoid it was found to consist of one large cavity filled with pus. The antrum was located high up and well above this cavity, and appeared as though it had not been opened at either of the previous operations. Surrounding the antrum there was a good deal of sclerotic bone. This was removed, and the epitympanic ring of bone was broken down. Too vigorous sponging on the part of an assistant resulted in the dislocation of the short process of the incus. The attic was curetted internal and external to the malleoincudal body; the ossicles were otherwise not disturbed. The drum was left in position, and the operation was concluded in the usual manner, except that the posterior wound was left open at the inferior angle.

The important features of this case are: First, the excellent hearing at present—acoumeter at fifteen feet—despite the previous dislocation of the incus; second, that the ear is dry and has been so for three months.

Case 3.—F. C., nine years old, was admitted to the New York Eye and Ear Infirmary for the modified radical operation fifteen months ago, with the following history: The affected ear had been discharging for five years. One year previously a simple mastoid had been performed on the same ear by myself, and the wound had never completely healed.

Operation.—On opening the mastoid the antrum was found to be filled with cholesteatoma, which was removed. The usual modified radical procedure was proceeded with.

The features of this case to which attention is especially directed were: First, that within one year after a simple mastoidectomy had been performed a large cholesteatomatous mass had formed in the antrum, which was removed at the second operation. Dr. Blackwell said that previous to this he had believed that this product of chronic bone inflammation required a much longer period for its formation; second, despite the presence of cholesteatoma in the mastoid, the ear was now dry, and had been so for the past nine months.

Case 4.—M. McB., a young adult, was admitted to the New York Eye and Ear Infirmary fourteen months ago with a history that the ear had been discharging for the past fifteen years, following an attack of measles. The patient said that for the past three weeks she had suffered with the most terrific headaches, which prevented her from sleeping. The pain radiated from the affected ear to the frontal and occipital regions.

Aural examination on admission showed a moderate amount of foul pus in canal, a slightly fluctuating discharge in the fundus, no mastoid tenderness. There was a large perforation in the drum, occupying the lower and anterior two-thirds of the membrane. Shrapnell's membrane was intact, together with the upper half of the handle of the malleus.

Operation revealed a very hard cortex, a small celled diploeic mastoid, no free pus in the cells, a large space in the antrum region filled with granulations and pus. These were removed, and the internal and the external attic were curetted. The ossicles were not disturbed.

The hearing before the operation was: Acoumeter at three feet, moderate whisper at six feet.

After operation the hearing was about the same.

The interesting features of this case are: First, the terrific headaches, which were relieved by the operation; second, there was no marked inflammation found in the mastoid or meninges to account for them; third, while the patient still has a wet ear, the discharge is much less and decidedly of a tubal character, and is not noticeable to the patient; fourth, with a healed antrum and mastoid cavity, the patient is insured against the development of serious intracranial complications, despite the presence of a wet ear; fifth, the patient's previous good hearing was not impaired by the operation.

SUMMARY.

In the first two cases the superior portion of the epitympanic ring of bone was removed as shown by Plate II.

In the last two cases the epitympanic ring was left in position, e. g., Plate I.

All of these were cases of chronic running ears. Three of them are now dry and have been so from three to nine months. In three cases the hearing has been improved since operation; in the fourth case it has remained about the same.

In dressing these cases it is most important to prevent a thick plug of granulations from developing in the attic and antrum regions, as it easily becomes infected and serves as a constant source of infection to the entire wound. The most satisfactory method of preventing this is to remove all packing from the canal at the end of a week—even removing the cotton plugs from the meatus—permitting the atmospheric air to freely enter the interior of the wound. This, combined with a system of cleansing the cavity, which the patient himself can carry out at home, has proved very effective.

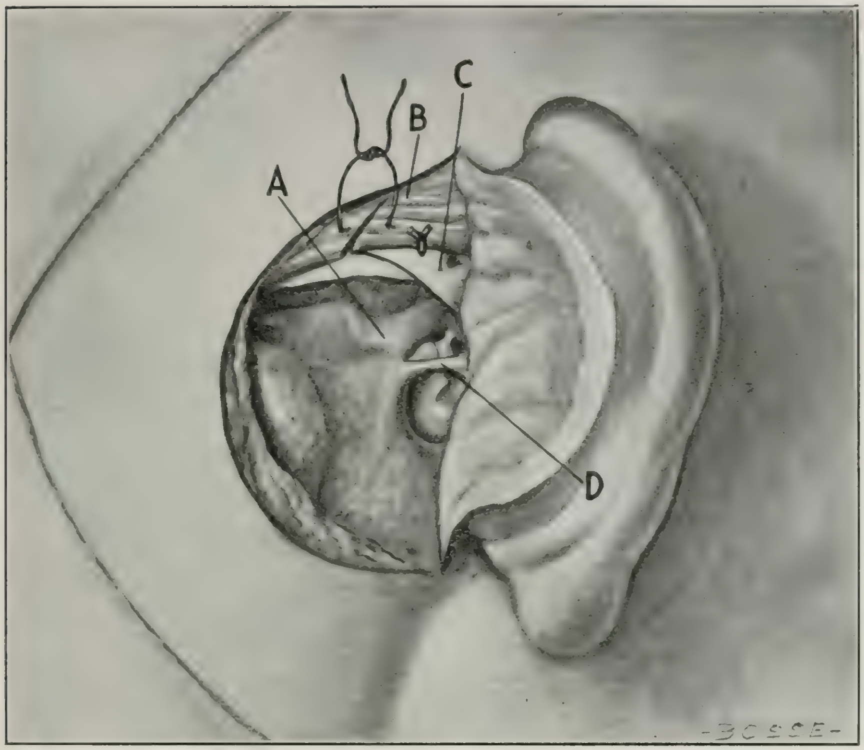


PLATE I.

View of operation prior to closure of posterior wound. Epitympanic ring of bone has been left in position. Drum and ossicles are intact. A—External semicircular canal. B—Temporal muscle. C—Plastic meatal flap sutured to fascia of temporal muscle. D—Epitympanic ring of bone, showing the malleoincudal body lying just above and internal, and the drum below.

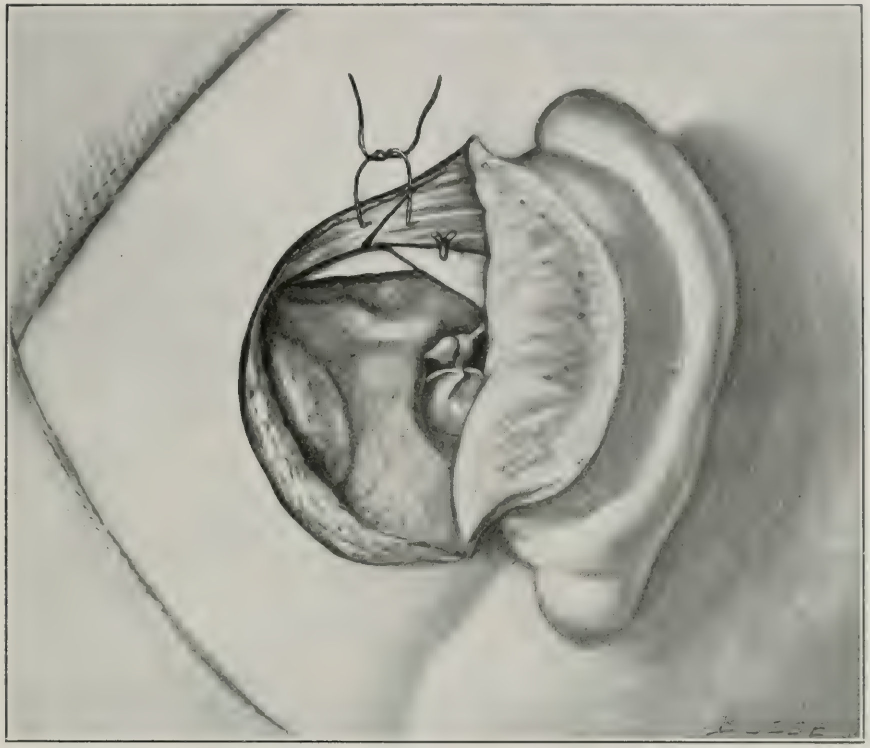


PLATE II.

View of operation completed, when the epitympanic ring of bone has been removed in the superior quadrant. Drum and ossicles are left in position.

XI.

THE TONSIL QUESTION IN CHILDREN.

By G. W. Boot, M. D.,

Chicago.

The indications for tonsillectomy have widened so much since the advent of the focal infection theory that nowadays the tonsils are removed for almost any pathologic condition, and at times apparently simply because they are present. The operation has become so popular that it is recommended for all sorts of conditions. Everyone is doing tonsillectomy, and many times it is done, not only with no advantage to the patient, but even to his positive detriment. This is particularly true in children. Everyone doing dispensary work has repeatedly had patients brought in for tonsillectomy because "he has tonsils." Like oophorectomy, the craze for tonsillectomy has run wild; and now seems to have reached its height. It is to be hoped that from now on there will be less indiscriminate operating and more skillful work done.

Until the function of the tonsil is more definitely determined, we will not be able to decide as sharply as we would like just what tonsils should be removed and what should not. Better means of determining the presence and kind of infection is much needed.

There is no doubt of the fact that the tonsils are more active in childhood than in later life. If a tonsil is normal up to the age of puberty, it becomes so largely fibrous that in adult life it is of little or no importance functionally. The younger the child the more active the tonsil, and the more we should hesitate about advising tonsillectomy. The ease with which the tonsils enlarge during any infection of the throat in children shows them to be very active. The fact that many of these enlargements will subside with proper attention seems to be largely overlooked by the general practitioner, and at times even by the men devoting themselves to children's diseases. If a child with acutely enlarged tonsils is put on syrup ferri iodidi for two or three weeks, a wonderful reduction in size often follows, and many children with enlarged tonsils, but with no cheesy plugs or pus in the crypts, should be so treated before deciding that tonsillectomy is needed.

Anyone who has seen many throats that have been operated upon knows that deformities of the throat may follow even a tonsillectomy that seemed perfect at the time of operation. As for the tonsillectomies that were poorly done, the less said the better, were it not for the poor victims. We have all seen deformities of the velum, distorted arches, adherent pillars, absence of uvulas, infected stumps, and the like. I once even saw a cleft palate made by a tonsil operation. Naturally, such gross deformities are made chiefly by the inexperienced and occasional operator. These deformities not only are bad from a cosmetic point, but they may cause rhinolallia aperta, difficulty in swallowing, and so on.

The effect of tonsillectomy on the voice is of importance in children, though they are probably less affected than are adults, for nature has a wonderful adaptive power in early life, and children learn to modulate their voices, even though difficulties are present.

The question is often asked, what effect removal of the tonsils will have on the patient's growth. There seems to be a popular idea that removal of the tonsils influences the sexual development of the child. I have never seen any such result. Some of the children on whom I have done tonsillectomy have become unusually well developed physically. None, so far as I know, have had their development retarded thereby.

We are often asked to remove tonsils in children because of habitual colds. Unless the tonsils are so large that they obstruct the nasopharynx, or unless there are other reasons for their removal, I discourage tonsillectomy in such cases. It has not been my experience that removal of tonsils influences head colds, unless they are large or plainly infected. Removal of adenoids in such cases is another problem.

In cases of tuberculosis of the cervical glands it is a mistake to remove the infected glands and leave the tonsils. In such cases the infection undoubtedly enters through the tonsil, and removal of the tonsil is even more important than removal of the glands. The tonsils in such cases will often, if not usually, be found to contain a mass of rotting, cheesy material. Tuberculosis of the cervical glands seems to be on the decrease since tonsillectomy has become popular.

In chronic tubal occlusion the tonsils are usually quite as much at fault as the adenoids. In such cases the tonsils are apt to be of the buried type, and on superficial examination may not seem to be at all enlarged. Pressure on the outer side of the tonsil by means of the examining hook will show that it extends well up into the velum. Many cases of chronic suppuration of the middle ear are favorably influenced by tonsillectomy. On the other hand, acute suppurative otitis media sometimes follows on tonsillectomy.

Streptococcus infections justify removal of the tonsils more often than other types of infection. If a child has a nephritis due to a streptococcus infection of the throat, the operation is indicated if the patient's general condition warrants the risk. The same is true of endocarditis; though in endocarditis it is well to wait for the subsidence of the acute symptoms.

If the tonsils can be definitely shown to be infected, their removal on account of rheumatism is indicated. At times removal is indicated in the absence of definite evidence of infection, provided that other sources of infection are excluded.

It is questionable whether tonsillectomy should be done to relieve chorea. If the tonsils are definitely diseased, remove them. If not, I would hesitate, for I have repeatedly seen tonsillectomy done in such cases without effect on the chorea.

I expect the next generation to see less otosclerosis than the present one, because so many tonsillectomies have been done in children. This disease closely resembles a disease known to be the result of a focal infection, and furthermore has its advent in childhood and adolescence. If it is a focal infection, as I believe, removal of the most common source of infection in childhood should have a marked influence on its development, just as tonsillectomy has influenced the development of tubercular cervical adenitis.

The tonsils are but part of the lymphoid tissues in the throat. There are many islets of lymphoid tissue scattered over the pharynx, and especially behind the posterior pillars. These islets seem to be particularly prone to compensatory enlargement if the tonsils are removed in early childhood. The condition that results is much like chronic granular pharyngitis from any other cause. This is one of the disadvantages that should be kept in mind in recommending tonsillectomy in children.

An occasional death from hemorrhage in a bleeder should remind us that we ought always to inquire into the existence of this condition in children, and if there is any doubt, to make a coagulation test of the blood before operation. This is so easily carried out that the few minutes spent in doing it are well repaid by the certainty the operator feels in a case where there is supposed to be a tendency towards bleeding.

Reports of postoperative pneumonia and lung abscess have become so frequent that this danger should be considered, as well as a means for its prevention. In most cases the cause will be found in too deep anesthesia and improper position of the patient, with resulting inhalation of infectious material from the throat. It cannot be insisted upon too much that general anesthesia for tonsillectomy should not be carried out as for ordinary operations of general surgery. The less anesthetic the patient gets, if he is insensitive to pain, the better. The anesthetic should not be pushed so far that the laryngeal reflex is abolished. If this is remembered the patient will not inhale infectious material, or if he does, he will immediately cough it out before it can reach the deeper parts of the respiratory tract to set up pneumonia or lung abscess.

Rapidly acting anesthetics like ethyl chlorid, ethyl bromid and nitrous oxid are not suitable for tonsillectomy because of their evanescence, and because to put the patient to sleep with them long enough to do tonsillectomy the laryngeal reflex must be abolished.

The anesthetist who insists on putting the patient soundly to sleep and the operator who wishes the patient so dead to the world that he can dissect out both tonsils without additional ether, are the ones who may expect lung abscesses. No one has the moral right to do tonsillectomy if he keeps his patient under a general anesthetic an hour or longer.

Anesthesia in the erect position or on the back, because it facilitates inhalation of infectious material, is to be condemned. The suction apparatus is cumbersome, adds one more instrument to fill up the small working space, and tends to make the operator careless about the amount of time he takes and the amount of anesthetic he uses.

Cheesy plugs are not so common in children as in adults. If they are present together with evidence of focal infection, one is justified in removing the tonsil.

Peritonsillar abscess always and tonsillar abscess when it can be diagnosed, justify tonsillectomy. The former is so apt to recur when once the peritonsillar tissues have become infected, and the condition is so distressing and at times dangerous, that I make it a rule to advise tonsillectomy.

Hypertrophy, the classic indication for tonsillectomy, may or may not require the operation. If the tonsils are so large that they obstruct the nasopharynx, remove them. If they are the projecting kind that on cursory examination seem large simply because they are all seen, and no part is hidden behind the anterior pillar or in the velum, then it matters little whether they are removed or not. These are the easiest removed, with the least resulting deformity, and ordinarily with no particular change in the child's condition. Such a tonsil is rarely the seat of a focal infection or the cause of tubal occlusion. It can be removed by any method with perfect satisfaction, and is the kind that leads the occasional operator to think he is capable of doing tonsillectomy.

Tonsillectomy is occasionally indicated in diphtheria carriers, though I have never done it for this cause.

There is no doubt that children who have had tonsillectomy done are less susceptible to the infections of scarlet fever, diphtheria and ordinary tonsillitis, though they are by no means immune to any of them, and no patient should have tonsillectomy done with the idea that he will never again have a sore throat.

The following are good rules to follow in doing tonsillectomy in children:

1. Operate only for definite disease.
2. Be sure the condition of the tonsil is the cause of the disease.
3. Always do a urinalysis before operating.
4. Always inquire for a possible history of bleeding.
5. If not certain, test the coagulability of the blood.

6. Don't push the anesthetic to the abolition of the laryngeal reflex.

7. Don't be slow in operating.

8. Don't destroy a functionating organ unless the gain more than offsets the loss.

9. The younger the patient the more carefully the need of tonsillectomy should be established.

XII.

CLINICAL PROBLEMS RELATING TO THE FAUCIAL TONSILS IN ADULTS.

By George E. Shambaugh, M. D.,

Chicago.

The treatment of the faucial tonsils in adults has until quite recently consisted for the most part in the care of cases during an acute attack of tonsillitis. Very little was attempted in the treatment of chronically infected tonsils or for the prevention of recurring attacks of acute tonsillitis. Two causes prevented more being attempted in the treatment of these conditions. One was the failure on the part of the profession to appreciate the serious menace, either from the occurrence of acute tonsillitis or from chronic infection in the tonsils; the other cause was the unsatisfactory methods at our command for the treatment of tonsil trouble in adults.

The general physician, as well as the throat specialist, has only recently begun to appreciate the serious systemic conditions which owe their origin to infected tonsils. It is now generally recognized that such conditions as neuritis, acute and chronic rheumatism, Bright's disease, and cardiovascular degeneration owe their origin for the most part to foci of infection such as are frequently found in the faucial tonsils. Evidence is accumulating, moreover, which throws suspicion more and more on the tonsils and other foci of infection as the cause of gastric and duodenal ulcer, various lesions involving the eyes, as well as acute and chronic neuritis of the eighth nerve.

With the development of this knowledge regarding the part which acute and chronic infection in the tonsils takes in the etiology of systemic disease, there has naturally arisen a demand for effective methods of treating infected tonsils. The older methods, such as slitting the tonsil pockets and cauterizing the tonsils, were so often unsatisfactory, that we were frequently inclined to leave the tonsils entirely alone rather than attempt this treatment. It has only been since we have learned that tonsils in adults can be safely enucleated that we have found a satisfactory method of handling tonsil trouble in adults.

Starting, then, with the assumption that chronic infection in the tonsils, as well as the tendency to attacks of acute tonsillitis, is often a distinct menace, especially as the cause of serious systemic disease, and with the conclusion that enucleation of the tonsils is the proper method of treating tonsils suspected of causing systemic infection, I wish to discuss some of the clinical problems which confront us in the treatment of tonsils in adults.

We have heard a good deal in recent years about the practice of indiscriminate tonsillectomy, the socalled "massacre of the tonsils," especially in children. Those of us who have been most interested in the question of the relation of tonsil disease to systemic infection are especially anxious that the proper indications be recognized before the removal of the tonsils in adults is advised.

The statement is sometimes made that tonsils in adults are never found to be normal. Even if we accept this statement, we must, nevertheless, insist that the indiscriminate removal of tonsils, even in cases suffering from systemic disease of focal origin, is not indicated. We are free to admit that the indication for tonsil removal may at times be quite obscure, but we cannot emphasize too strongly the fact that the mere presence of a systemic infection is not of itself an evidence against the tonsil. In order to avoid as far as possible unnecessary operations on the tonsils, it is of great importance that the throat specialist should not assume a greater responsibility in the management of such cases than he is competent to handle. There are many cases in adults where the indications for tonsil removal rests chiefly on the local trouble in the pharynx. In these cases the throat specialist is, as a rule, best able to decide what should be done. Cases coming under this head include those where there is a tendency to attacks at frequent intervals of acute tonsillitis. In a few cases the local annoyance from the accumulation in the tonsil pockets of foul-smelling cheesy concretions, especially when the tonsils are much enlarged, is sufficient to constitute a proper indication for removal of the tonsils. In the great majority of cases in adults where the removal of the tonsils is indicated, the question is not so much one of local annoyance to the patient as it is one of systemic poisoning from chronic infection in the tonsil, or as the result of acute attacks of tonsillitis. It is in just these cases where the cooperation between the throat specialist and the internist is necessary, if we are to avoid the risk of doing unnecessary operations.

The cases are relatively few where the development of systemic trouble, such as rheumatism, Bright's disease, or endocarditis takes place simultaneously with an acute attack of tonsillitis, so that the throat specialist can conclude, without hesitation, that the tonsil infection has been the cause of the trouble. In the great majority of cases where the tonsils are responsible for the systemic infection, this conclusion can be reached only after an exhaustive examination of the case, which can be done more efficiently by the internist than by the throat specialist. In many of these cases where the tonsils are suspected of causing systemic infection the first thing to be determined is the exact nature of the trouble from which the patient is suffering, and whether this trouble is one which is known to be the result of focal infection. The throat specialist ought not to assume the responsibility of passing on these questions. They are problems which fall more properly in the work of the internist, and often constitute one of the most difficult problems for him to solve.

When it is determined that a patient's trouble is probably the result of a focal infection, the next step is to determine the probable location of this focus which is causing the systemic trouble. Here again the problem is more one for the internist than it is for the throat specialist. The examination of the tonsils and of the nasal accessory sinuses is but a small part of the work to be done before the probable focus can be determined. Not infrequently several suspicious foci of infection are discovered, and the question as to the best course to pursue in clearing up the several foci is again one for the internist, who is in a better position to view the case from all sides.

The responsibility which belongs peculiarly to the throat specialist is in determining whether the tonsils are the seat of an infection which might be capable of causing systemic trouble. The answer to this question in some cases can be readily decided, but in others the answer is not so apparent. When, for example, there is a distinct history of recurring attacks of acute tonsillitis, we are quite justified in concluding that the tonsils are the carriers of infection which may be responsible for a systemic trouble. Not infrequently there is no distinct history of acute tonsillitis, but the patient states that there are frequent attacks of slight sore throat. In such cases the sore throat, as a rule, is nothing less than mild attacks of tonsillitis. It is usually best, however, to ask the patient to return during an acute attack, when an examination of the tonsils can determine whether they are the seat of an acute inflammation.

In many cases an examination of the tonsils discloses unmistakable evidence of chronic infection. In not a few cases pus can be actually expressed from the tonsil. More often evidence of infection consists in a congestion involving not alone the free surface of the tonsil, but of the anterior pillar in proximity to the tonsil. Enlarged tonsils from which masses of epithelial débris can be expressed from the crypts should always be regarded with suspicion. On the other hand, tonsils which are the seat of a few cheesy plugs, especially when they are not associated with a distinct congestion, can hardly be classed as infected tonsils from the local findings alone.

It is a well known clinical fact that tonsils which are the seat of such frequent attacks of acute inflammation as to justify the conclusion that they are persistent carriers of infection may, nevertheless, between these acute attacks, show very little that could be construed as evidence of chronic infection. It is also a very frequent experience to discover distinct evidence of chronic infection in tonsils; as, for example, the presence of pus which can be expressed from the tonsil, where there has been no history of attacks of acute tonsillitis or of sore throat. It is evident, therefore, that there may be cases where neither from the history of acute attacks of tonsillitis or from the local findings in the tonsils is there any positive evidence of tonsil infection, and still they may be the seat of an infection capable of producing the most serious systemic trouble. It is this class of cases where the indication for removal of the tonsil is the most difficult to work out. Because such cases are known to occur, must not be construed as an indication for indiscriminate tonsillectomies in cases suffering from systemic infection. Only in cases where the systemic infection is of a serious character, and where a careful search by a competent internist has failed to discover any probable focus of infection, should the advice be given to have the tonsils removed.

The operation for the removal of tonsils in adults is by no means a minor operation, and while the indications for this operation have been greatly increased by a better appreciation of the menace existing in tonsils the seat of chronic infection, still it is quite evident that the indiscriminate removal of tonsils in adults is to be discouraged. The decision to remove the tonsil can be reached in a great many cases only after a careful investigation, which, to be complete, requires the cooperation of the internist.

XIII.

THE SURGICAL TREATMENT OF SUPPURATION IN THE JUGULAR BULB.

By John McCoy, M. D.,

New York.

In a small proportion of cases of thrombosis of the lateral sinus and jugular vein, in which we have performed the operation of incision of the sinus, excision of its outer wall and ligature, or excision of the jugular vein, we are confronted with a condition of suppuration from the jugular bulb with septic symptoms. This is evidenced by the persistent appearance of pus welling up at each dressing from the lower end of the sinus; in other words, a small quantity of pus wells up from the jugular bulb into the lower portion of the mastoid wound.

A typical case which occurred in the writer's practice was as follows:

A boy, aged ten years, was seen in consultation, and gave the following history: Two weeks previously had earache, followed by discharge from the right ear. About three days before the writer saw him he had a chill, fever and sweat. This recurred on each succeeding day, and when seen he was found to have a purulent discharge from the right ear through a small sized perforation in the lower part of the drum. He was slightly tender over the mastoid antrum and tip. A blood culture was immediately taken, and the following day disclosed the presence of streptococcus in the blood. The boy was operated on that day, and the mastoid showed a diploeic bone, and the cells were congested and filled with granulations. The sinus plate was found intact and hard. The sinus was uncovered and found discolored and covered with a thin layer of exudate. It did not pulsate and felt firm to pressure. It was opened and found completely filled with clot. No flow from either torcular or jugular end. The outer wall of the sinus was removed and the clot curetted out. The clot extended far back to the torcular. The clot towards the bulb was not disturbed, but the neck was opened and the internal jugular vein was resected from one inch above the clavicle far up towards the bulb. No clot was found in the vein. It was slightly discolored and sheath adherent.

The child stood the operation very well, and after zig-zag temperatures, from 101° to 104° for a week, the temperature gradually dropped to 99° in the morning and 100½° in the evening. After running along this way for five or six days the temperature again became septic, ranging from 100° to 103°, and the few drops of pus which welled up from the lower cut end of the sinus of the mastoid wound gradually increased to a half teaspoonful or more at each daily dressing. The child became increasingly restless and sleepless, and batted his head about a great deal. After a week or so of these symptoms the writer decided that it would be the wisest plan to secure adequate drainage from the jugular bulb. It was at first thought that he might be able to pass a fine catheter containing a fine wire from the lateral sinus into the bulb and down through the vein. This, however, was found quite impracticable, and with the assistance of the general surgeon the neck was opened and the jugular bulb was reached with considerable ease. The vein was split up and the bulb was drained (according to the method later to be described as Tandler's).

For the relief of this condition several surgical procedures have been devised. This paper is based on a series of dissections which have had for their basis a determination of that method which accorded the easiest and safest way of draining the jugular bulb and avoiding injury to contiguous vital anatomic structures.

Of the several methods devised for relieving this condition, the writer will describe some briefly, because they do not appeal to him as surgical measures worthy of serious consideration, and others more in detail, because they might be employed with more or less success. He will then give his conclusions as to the best of the latter.

I would first mention the skin fistula of Alexander. The skin fistula method of Alexander is probably the simplest measure devised for drainage of the bulb. It consists in suturing the jugular vein, where it has been cut off, high up in the neck, to the skin incision in the neck, leaving the vein open. This would probably prove a more or less effective method if we found pus welling up from the bulb at the time of operation, but if this condition did not exist, then we would render our patient liable to a hemorrhage from the inferior petrosal sinus.

Neumann has devised a method in which he says that he passes a Gigli's saw over which a Nelaton catheter has been passed, from the sinus at the lower end of the mastoid wound through to the bulb, until it comes out at the peripheral end of the jugular; or reversed, the instrument may be carried from the stump of the jugular into the bulb. After the introduction of the instrument, the catheter is removed and the wedge of bone surrounding the bulb may be sawed through from within outward. This method the writer considers very difficult, if not impossible, in the majority of cases.

Piffl has devised an operation in which, after separating the membranous canal wall, as we do in the radical mastoid operation, and pushing aside all tissues up to the glaserian fissure, by means of bone forceps he removes the floor and part of the anterior wall of the bony external canal to the hypotympanum. He says that having gotten this far, as a rule, the upper portion of the internal jugular vein makes its appearance, then exposes the bulb by exposure of its outer margin. This the writer considers a most difficult and dangerous procedure.

Voss has devised an operation in which he enters the bulb from the lower portion of the mastoid wound. The steps are briefly as follows:

(1) After exposing the sinus and removing the overlying bone downward to the lowest part of the vein, the vein becomes more and more horizontal, the lateral wall of the sinus becoming the inferior. (2) This lower wall of the sinus is followed by removing bone with concave gouges. (3) By following the course of the vein with a probe, the distance of the anterior wall of the jugular fossa can be determined. (4) The next step in the operation, the exposure of the bulb, is accomplished by very cautiously removing the bony wall above

the anterior boundary of the jugular fossa (to avoid injury of the horizontal semicircular canal the bone is not removed higher than one-half centimeter). He says the posterior membranous wall of the bulb is readily exposed in most cases of a well defined bulb. After the posterior wall has been removed up to the roof of the bulb, it sometimes becomes necessary to remove the edge of bone which marks the separation of the sinus proper and the bulb. This shows the sharp knee from where the lateral wall of the sinus goes into the posterior wall of the bulb.

The method of Grunert is more extensive than this, and may be described as follows:

(1) The complete resection of the tip of the mastoid. (2) The separation of the three muscles from the base of the skull, namely, the sternomastoid, digastric, and the splenius capitis. (3) The soft parts are retracted as we work toward the base of the skull in a horizontal direction, in order to reach the jugular foramen. (4) The operative field is sometimes considerably narrowed by a prominent transverse process of the atlas. This may have to be resected. In doing so one must be careful not to injure the vertebral artery. (5) The rectus capitis lateralis must now be pushed aside in order to remove the last bridge of bone between the free margin and the jugular foramen. (6) The bulb is now split from the sinus end, and the free lying lateral wall is excised. (7) The bulb is now inspected and curetted free from thrombus masses. If the internal jugular appears normal, he stops there and packs the bulb with iodoform gauze, but if the vein shows a thrombus, then it can be laid open and packed.

The Tandler operation is as follows:

(1) The mastoid incision and the incision for the jugular are converted into one. The sternomastoid muscle is separated for its entire length and pushed posteriorly. (2) You can now search for the spinal accessory nerve. When it is found, it is tied loosely with a suture so that it may be in good view during the operation. We may dissect without danger and follow this nerve almost to its exit at the jugular foramen. (3) If now the finger is passed deeply into the wound, we find a space between the styloid process and the mastoid process. Here the exit of the facial nerve through

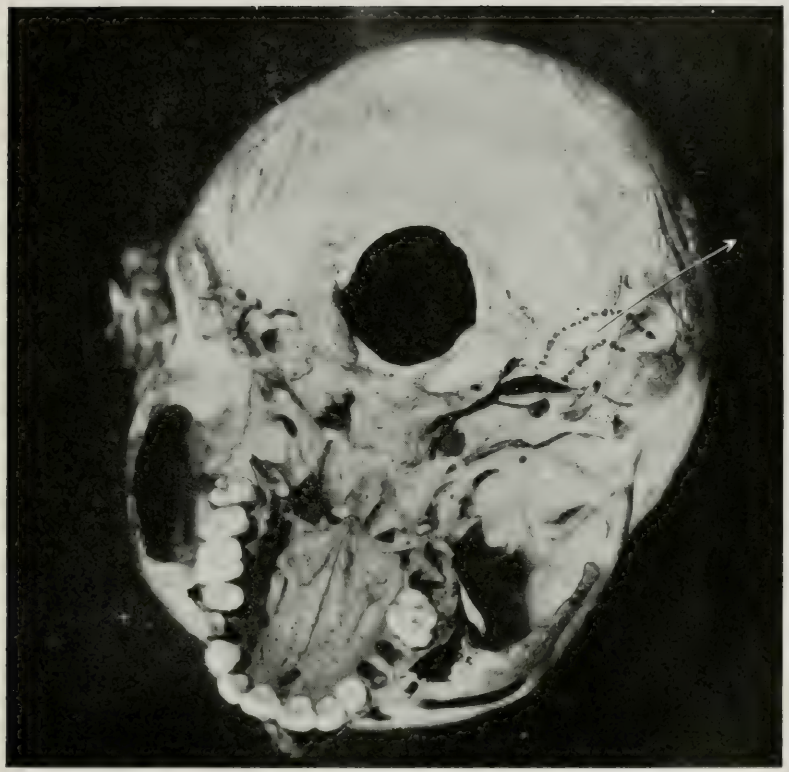
the stylomastoid foramen may be exposed. (4) The digastric muscle is separated from the digastric fossa and shoved forward and downward. If we work on a line below the stylomastoid foramen and this muscle, there will be no danger of injuring the facial nerve. (5) After pushing aside the digastric, the occipital artery may be ligated in two places and cut. (6) The jugular vein may now be separated with the margin of the jugular foramen, which can be felt with the finger. Now loosen the uppermost portion of the vein, after pushing aside the periosteum at the base of the skull, and the rectus capitis lateralis. (7) The bone is now removed from the sigmoid sinus to the bony margin of the jugular foramen, and the sinus bulb and vein are split wide open.

Several anatomic facts must be borne in mind in the performance of these operations. In the first place, it is found that the jugular bulb is very deficient on the left side of the skull in the majority of people (some say as high as seventy-five per cent). This is shown in the skull pictured; also the fact that three large and important nerves pass through the jugular foramen, namely, the glossopharyngeal, the pneumogastric, and the spinal accessory. In our manipulations we must be careful not to disturb or injure these nerves, as the spinal accessory sometimes is situated ventrally and sometimes dorsally. This is the nerve which is most apt to be injured.

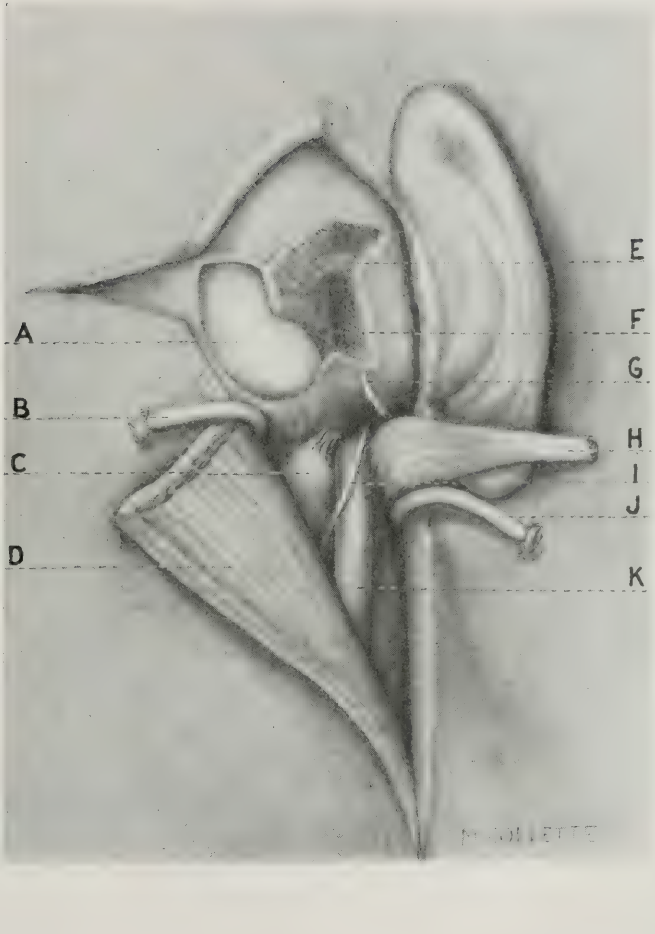
Grunert gives as indications for the bulb operation the following:

(1) For opening of the bulb at the primary operation, isolated cases of both mural and obstructing bulb thrombosis. (2) Cases where diseased bone extends to the jugular foramen, making necessary an exposure of the bulb. (3) Cases of otogenic pyemia with a sinus and bulb thrombosis, with severe septic symptoms. Secondary opening of the bulb is indicated after freeing the sigmoid sinus and performing jugular ligature or resection, when severe pyemia exists, high fever, chills and suppurative destruction of the clot in the bulb.

In ending, I may state that the operation described by Tandler gives the easiest, simplest and safest method of reaching the jugular bulb, and most skillfully avoids danger of wounding the surrounding vital structures.



This illustration shows a well developed jugular bulb on right side, and a very poorly developed one on left side. Area within dotted lines shows route from mastoid to jugular bulb.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XIV.

SALIENT FACTS REGARDING TONSILS IN CHILDREN AND ADULTS.*

By Joseph C. Beck, M. D.,

Chicago.

It would appear that another paper on the tonsil would be superfluous before this society, especially after two such splendid contributions to the subject as were presented at the last meeting. Perhaps you will find it superfluous, but to the writer, as well as a number of gentlemen present at that meeting, it appeared that the matter of the importance of the tonsil in the human economy was not sufficiently covered and an impression was left in the minds of many that everyone needed an apology to himself and his patients on whom he had operated for the removal of their tonsils. After listening to the two papers and some of the discussion the writer realized that the time limit allowed for discussion would not permit him to bring out the points he wished to emphasize, and consequently this paper is presented.

I shall ask your indulgence in this presentation by overlooking the unsystematic manner in which it is done, simply because it will contain mostly casuistic reports.

As to indications for operation—that is, tonsillectomy—whether in children or adults, I wish to make a broad statement, that with the exception of a few contraindications, every tonsil is better out than in, and I have no knowledge of a single instance where the patient was worse off from the tonsillectomy than he would have been with the tonsils not removed. This refers to tonsillectomy and not tonsillotomy. These contraindications I admit are:

1. All acute inflammations or infections.
2. Luetic, particularly ulcerative processes.
3. Advanced tuberculosis.

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*Read before the Chicago Laryngological and Otological Society, December 19, 1916.

4. Advanced cardiovascular changes; that is, in individuals who would be endangered by any sort of a surgical procedure, as in all constitutional diseases where the anemia is very marked; for instance, leukemia, pernicious anemia, etc.

5. Advanced cases of diabetes mellitus.

6. Low coagulating power of the blood, as in true hemophilia, cholemias, etc.

7. Blood pressure over 225 systolic.

8. Infants below one year of age.

9. Grave mental diseases in which marked excitation is present, as Graves' disease, Basedow's disease, maniacal state, etc.

10. And, as one of my students said in answer to this question, anyone that has never had sore throat, and is in perfect health as shown by physical and laboratory examination.

This is an ava rara. I am cognizant of the fact that tonsillectomies are, have been, and perhaps will be performed in many of the above mentioned conditions as contraindicated, but I should advise against such procedures. That tonsillectomy is not overdone or a fad is proven by the mass of evidence of the wonderful results, and the time limit has long passed in comparison with other surgical procedures, which had only a temporary existence, such as uvulectomy, inferior turbinectomy, certain eye muscle operations, ovariectomies, and the oroficial surgery.

Tonsillectomy, like appendectomy, cholecystectomy and a few others, is here to stay, and an effort on our part to stop this splendid life work in making a stronger, better race, would be a discredit to our profession. It should be our purpose to improve our technic so as to do away with any and all possible dangers and after-effects from this operation.

Some of these were brought out in the papers and discussion, but I wish to take issue with some and perhaps add others not mentioned.

One bad result reported is that of scars or deformities causing trouble with voice production. Dr. Kenyon would like to see an intracapsular tonsillectomy in cases of singers. I have never operated on a prominent singer, but have on a fair number of good professionals and amateurs, and I have never seen any bad effect nor heard complaints following a carefully performed tonsillectomy with the capsule. I have developed, however, an operation (intracapsular tonsillectomy) which I explained to Dr. Kenyon, and invited him to see. The description of this technic will follow later. Following the contraction of these scars, there have been complaints of pains in the throat, but these were not lasting.

Another bad effect of the tonsillectomy, especially when radical, refers to symptoms resembling cretinism and marasmus in infants up to the third year; consequently, with the rarest exception do we operate under that age. This observation as well as age limit is not conceded by many laryngologists, but we have observed it often enough to make us adopt such a course.

Another after-effect following tonsillectomy, as in any other operation, is the postoperative neurosis, especially in adults; but since we have adopted general anesthesia this does not occur so often. My method is as follows: Gas, followed by ether, rebreathing or drop method, until relaxation is sufficient to permit introduction of the mouth gag; then this is followed by the ether vapor method, during which the major part of the operation is performed. This is followed by the open gas method, to completely control any oozing, especially from the adenoid region. Patient should awaken on the operating table and return to bed with a dry throat. The laryngeal reflexes are never allowed to become abolished for a second. As the pupillary reflexes are always the indicators of the depth of anesthesia, the pupils are never allowed to become dilated. Therefore, the patient is neither superficially nor deeply anesthetized—just enough to have the pharyngeal muscles completely relaxed and be unconscious.

Another matter of importance is the prevention of aspiration of blood which may cause abscess of lungs or pneumonia, the subject that has recently been very prominently brought before the profession, especially by the general surgeon. This can always be prevented by employing suction and immediate and permanent control of the bleeding. We have never had a case of either abscess or pneumonia following tonsillectomy. There is not any doubt that the best way to deal with bleeding vessels is to grasp them and tie them, and this we do in every case after we have locked a sponge in the tonsil cavity for a few moments, if we find that the bleeding has not stopped. It is, however, extremely rare that we have to resort to tying. The quickest and surest way of tying a vessel in the tonsillar fossa, especially at the inferior pole, is to grasp the bleeding point with an Ellis pick-up forceps, which is specially constructed for this purpose, and which permits some tissue to be lifted, below which it is easier to tie. A needle may carry the ligature about this grasping forceps to further prevent it from slipping after it is tied. To prevent subsequent oozing, we have in the past two and one-half years employed in every case of adults (sixteen years and over) a small inlay of gauze or rubber, stitched over by the edges of the anterior and posterior pillars. This is allowed to remain from two to twelve hours. This procedure is employed in preference to ligation, because it is easier, although perhaps not as agreeable to the patient. This control of the bleeding will prevent another very annoying condition, namely, the swallowing of blood, with subsequent vomiting and kapremia.

Another disturbing factor following tonsillectomy, especially dissection, is the secondary reaction of exudate formation and infiltration. The use of iodin—and we prefer balsam of Peru in olive oil—will diminish this considerably. Marked septic cellulitis we have never had, but in some cases have had considerable edema of the uvula, perhaps due to the unnecessarily prolonged traction by the catheters which we employ in exposing the postnasal space. The frequent complaint of earaches, pain on swallowing, or a distinct nasal twang to the voice are all transitory and usually disappear within a week. Tincture of iodin, applied to the fossa, relieves these conditions, as will the gargling of weak warm tea and seltzer water, equal parts.

Another more rare condition following tonsillectomy, and, for that matter, any operation under long continued ether anesthesia, is acetonemia or acidosis. The routine use of bicarbonate of soda, thirty to sixty grains in one-half pint of water, introduced per rectum about one-half hour before operation, will go a long way toward preventing this condition, and if it occurs, characterized by considerable nausea and vomiting, with noticeable sweetish breath, it can be controlled by more bicarbonate of soda solution and the additional use of grape sugar.

One of the most disappointing results following tonsillectomy is the failure to obtain benefit from the operation, either from the local or more frequently general or distant conditions. For instance, it has occurred to us that following a perfect tonsillectomy the patient will have an attack of acute sore throat, and examination reveals a white spot or two. This is not tonsillitis, but a folliculitis in the lymphoid tissue, which in many instances has increased as a compensatory factor. Very frequently the lypmhoid tissue at the base of the tongue and back of the posterior pillars will undergo marked hyperplasia, but this does not last, and only in rare instances is it necessary to attack it surgically.

Glands of the neck, if they are tuberculous and broken down, will often not disappear following tonsillectomy. I do not consider the remains of tonsils as a bad after-effect from tonsillectomy, because it is not a tonsillectomy. It may, however, occur in the hands of the best operators, and when discovered, reoperation is necessary.

A hypertrophied plica tonsillaris which must not be mistaken for remains of a tonsil does no harm.

When a general condition, as articular or periarticular rheumatism, endocarditis, myocarditis or pericarditis, pleuritis, myositis, neuritis and perineuritis, nephritis with the characteristic blood picture of a chronic septic absorption, does not immediately disappear in every instance following tonsillectomy, there appears to be some skepticism expressed as to the value of the procedure. In most of these cases in due time, especially if aided by autogenous vaccination, either specific or haphazard, as well as the elimination of other foci, very satisfactory results are obtained. There are a fair number of patients suffering from these conditions in whom, no matter what is done, the symptoms persist. An alcoholic neuritis, a brachial plexus neuralgia from pressure of a supernumerary rib, a sciatica from pressure of a sliding sacroiliac joint, will not be influenced by a tonsillectomy. However, I have seen tonsils operated upon in such cases.

In conclusion I wish to mention some general conditions in which tonsillectomy appears to be of benefit, namely, in the disturbance of the glands of internal secretion. Hyperthyroidism is very beneficially influenced, and many cases of thyroidectomy for this condition are unsuccessful until the tonsils have been removed. Otosclerosis, which was early looked upon as an osteoporosis, due probably to disturbed function of the glands of internal secretion, a hypoadrenalism, is corrected by a tonsillectomy, which should bring such a case to a standstill. There is no scientific proof of these statements—only the clinical facts.

One of the most recent claims for the tonsils as a causative factor as an atrium of infection is in infantile paralysis. Rosenow and Nuzum have found in every case they examined a small diplococcus in the tonsils of these patients, which, when inoculated into rabbits, produced symptoms not unlike infantile paralysis. In Nuzum's work at Cook County Hospital, in all but two of the children that had had a tonsillectomy previous to the attack of paralysis, the disease was mild and complete recovery followed. These two had stumps of tonsils left. All the cases that had the tonsils removed subsequently to the disease made a more speedy recovery than those that did not.

So far as the pathology is concerned, we consider the surgical and nonsurgical forms. It is the former that we are mostly concerned in.

In 1902 I reported on my histologic studies of tonsils removed, and found there were varieties of changes, namely:

1. Hyperplasia.
2. Hypertrophy with inflammation.
3. Cicatrization with degeneration of epithelium in crypts, also known as caseation or cholesteatomatous formations (Pierce).
4. Chronic abscess formations (blind abscess or caseation).
5. Tuberculosis.
6. Sarcoma.
7. Carcinoma.
8. Actinomycosis.
9. Hyperkeratosis.

Since then I have found cases of cartilage in the tonsils, also stones within the crypts. My work includes histologic studies of two hundred and twelve individuals.

Each of these conditions has a distinct importance as to diagnosis and treatment, and would be of interest to go into more thoroughly, but there is no room for such consideration in this paper except to answer Dr. Libby that his classification of the pathology is inefficient. The terms submerged tonsils and peritonsillitis are very happily chosen, although neither is correct; that is, they do not exist as pathologic entities. Retracted tonsils and tonsilloperitonsillitis expresses more nearly the real condition. As to the bacteriology within the tonsils, many nonpathogenic varieties are found, and others which have nothing to do with either the local or general systemic conditions. They are, as a rule, not very active, but their toxins are positive in their production at least of blood changes. It is the slow and constant absorption of these poisons or toxins that is injurious.

I cannot close without expressing my deep gratitude, and I am sure that of all of you, to the men who have contributed so much to the tonsil question as Billings, Rosenow, Pynchon, Ballenger and Sluder. It does not matter what technic we employ in the removal of the tonsils, so long as we remove them all and nothing more.

Billings and Rosenow, in their development of the subject of focal infection, placed the tonsil among the most important agencies for the entrance of chronic infection. Pynchon was the pioneer of tonsillectomy, although his method by means of the electrocautery did not become popular. Ballenger perfected Pynchon's technic in performing tonsillectomy by dissection and snare, and Sluder originated a plan of tonsillectomy with the least amount of loss of pharyngeal tissue and traumatism, performing the operation with one instrument, without any preliminary dissection.

In an article published by me in the *Journal of Surgery, Gynecology and Obstetrics,* 1915, entitled, "The Evolution of the Tonsil and Adenoid Operations," all these factors were brought out in detail, but today I will satisfy myself by the above reference and the presentation (demonstration by lantern) of a collection which represent the main types of instruments in use from ancient times until the present day (Figure 1 to Figure 5).

By request I present our present method of dealing with the tonsils and adenoids.

Tonsillectomy.—Let me say at the outset that we always try to remove the tonsil with Beck's tonsillectome (original pattern), and if the dislodgement through the ring does not take place readily, we resort to the dissection rather than to persist in the ring technic. The steps are as follows:

(1) General anesthesia (preferably operating in the morning).

(2) General preparation. A light or no supper, and a mild cathartic the night before operation. In the morning a glass of milk or coffee or coca or nothing. The bowels should be washed out and a solution of bicarbonate of sodium injected into the rectum and allowed to remain.

(3) Atropin gr. 1/150 hypodermatically in adults, and in children, we give only 1/200 of a grain of atropin by mouth.

(4) The patient should be taken to the operating room on a cart, preferably without any information given to him in advance. The operating room should be quiet; the physician, whom the patient knows, the surgical nurse and anesthetist alone are present while the patient is going to sleep. Besides these three, one of the family may be present until the patient is unconscious.

A preparation or anesthetic room adjoining is preferable to the operating room for the administration of the anesthetic. When the technic of the anesthetic is so far advanced that it will be practical to do this, it will go a long way towards removing the terrors of an operation. In fact, if a patient could be put to sleep without knowing that he was to be operated upon at that particular time (something like Crile's idea of anocia association) many of the after-effects, as postoperative depression, etc., would be avoided.

(5) The patient is put in the recumbent position (not strapped to the table) until he is partially under the anesthetic. He begins by voluntarily breathing nitrous oxid gas from the mask, which is put on lightly until he shows evidences of slight cyanosis. The curved mouthpiece through which the properly warmed ether comes is put in the corner of the mouth, between the lips and teeth, under the gas mask. While the gas is being given the ether is supplied in slight quantity. The gas mask is now removed and either the rebreathing ether bag and mask (Figure 6), or a regular gauze covered

wire mask is substituted. As soon as the patient is sufficiently relaxed the writer's mouth gag (Figure 7) is introduced by the operator while the assistant passes the two small (size No. 2) urethral catheters through the nose into the oropharynx. The operator depresses the tongue and employs the suction tube to remove the mucous secretion, and then grasps the free ends of the catheter and withdraws them out the mouth. The assistant joins the two ends of each catheter by a spring clamp, to prevent their displacement during the tonsil operation. A gauze pad about the size and thickness of a hand, which is wet, is placed over the open mouth, and the flow of ether vapor is increased by the valve on the writer's anesthetic suction apparatus (Figure 8) so as to cause pharyngeal relaxation. The rhythm of breathing and the pupils are watched very closely, as the borderline between sufficient anesthesia and excessive narcosis is very narrow. We have never had a fatality or any other untoward result with this method of anesthesia, yet very deep anesthesia has occurred, and this is always unpleasant. The field of operation being thoroughly illuminated by the writer's head light (Figure 9) (nitrogen globe and aluminum reflector, which never gets hot), the right tonsil is first removed. With Beck's tonsillectome (Figure 10) (usually the medium size) in the operator's right hand the tonsil is lifted into the supratonsillar fossa until it appears like a small round mass behind the arcus palatinus. The instrument now lies diagonally across the tongue, in which position it is firmly held while the index finger of the left hand presses on the above mentioned round mass behind the arcus palatinus until it has passed through the ring of the instrument. The pressure exerted should be steady, and the counter pressure of the instrument should be equal to that of the finger. It is important to be sure that the entire tonsil has passed through the ring, particularly the upper pole, which can be engaged by giving the ring a sort of half twist. The entire circumference of the ring will usually be felt, if all the tonsil has been engaged. The index finger is held in this position to guard the tonsil from slipping back, the left thumb disengages the lock of the snare, and the right hand draws down the wire loop. The left index finger feels this wire under the soft palate as it is drawn down. The left index finger can now be taken away and the snare locked again. The tonsil is now observed everted from its capsule encircled by the snare, and at the lower portion of the anterior pillar a sort of a dimple is seen, which indicates complete eversion of the capsule. At this point it is necessary to decide whether to remove the tonsil with or without capsule. It is only seldom that we resort to the former or intracapsular method. The method is as follows: About one-half of the protruding mass is clipped off with curved scissors, then a sharp curette is used to remove the remainder. In all these cases the wire must be drawn down much more firmly, otherwise the capsule will slip back before all the crypt portion of the tonsil is removed. After the capsule has slipped back the field should be explored, and if an area is found suggestive of a tonsil crypt, it should be further curetted.

The extracapsular or usual operation is completed by grasping the protruding everted tonsil with a vulsellum forceps and the attachment severed by slowly turning the thumb ring of the snare. The tonsil is immediately placed into a sterile bottle for cultural study and for vaccines.

As soon as the tonsil is out the anesthetist grasps the jointed end of the catheter, corresponding to the side from which the tonsil was removed, and pulls firmly outward and backward, thus bringing the two pillars in close proximity and making a tonsillar pocket which retards the flow of blood sufficiently long to permit the operator to seize a gauze sponge with a Tuffier's or Ellis forceps and place it into the pocket, where it is held by the forceps grasping the two margins of the pillars (Figure 11). The size of the sponge is determined by the size of the cavity present after the tonsil is removed and the amount of bleeding. Great care should be exercised not to overfill the cavity, and in trying to lock the sponge in with the forceps to exert too much pressure and to injure the pillars. Any and all the blood that is escaping into the pharynx is removed by the suction apparatus guided by an assistant.

If in spite of the sponges bleeding continues for more than a half minute, the sponge is removed and attempt made to find the active bleeding point. By grasping separately the anterior and posterior pillars with Wild's forceps which hold well yet cause no injury, the entire cavity can be explored and the bleeding vessels easily caught by means of artery forceps. After a few moments they may be removed, and if the bleeding continues the vessel is tied. It is especially essential that this should be done when the vessel is in the lower pole of the fossa, under which circumstances it can only be observed when the base of the tongue is thoroughly depressed on that side. The bleeding point should be seized with an Ellis pick-up forcep and lifted up so as to permit a fine silk ligature to be placed around it. Long threads should be used and the technic which general surgeons use in ligating vessels deep in the abdominal cavity should be employed. If the ligature should slip off, it may be secured by passing a fine needle and thread about the forceps.

If bleeding still continues, the additional precaution may be used of placing into the cavity a piece of rubber sponge about one-third the size of the cavity. (Formerly we employed gauze saturated with compound tincture of benzoin to prevent putrefaction, but now we use the rubber sponge without any medication on it, since no odor is present when the sponge is removed after twelve to twenty-four hours.) A silk suture is placed through the margins of the two pillars and includes a particle of the sponge to prevent it from slipping out. It is best to put the suture at the lower third, tucking the sponge into the supratonsillar fossa, thus preventing it from slipping out.

The same technic is employed on the opposite side. The left hand should hold the instrument. Some find this a little difficult at first. If the operator insists on using the right hand, it will be necessary to put the patient in the Rose position and to stand behind the patient, when removing the left tonsil.

Having completed the tonsillectomy, the postnasal space is explored by having the anesthetist draw outwards and backwards on both the catheters. It stands to reason that the exposure of this area is not so good when the sponges are within the tonsil fossæ as when these are empty. In children that is the case, because the sponges are seldom used and the view of the adenoids is particularly good. Adenoids are often seen on the posterior surface of the posterior pillars, a position in which they cannot be seen except by such thorough retraction of the soft palate. These lateral masses are invariably first attacked by means of the ring curette (Figure 12), on account of the greater bleeding. The main mass of adenoids we usually take away in part with a Stevenson adenotome (modification of LaForce) and the operation finished with a Barnhill curette, employing a very gentle stroke and not attempting to remove the adenoids en masse, but rather in such small particles as will be sucked up and not obstruct the rubber tube of the suction apparatus. We always have a duplicate suction tube available in case a particle of adenoid tissue blocks up the tube. About this time a fair flow of nitrous oxid is given to the patient by the open method and the ether cut out. The remainder of the operation is done under open mouth gas anesthesia.

The nasopharynx being cleared of the adenoids, we press against this denuded area, which has often the appearance of a gouged out excavation, a firm gauze sponge mounted on an artery forceps. This is held there for a few moments and perhaps replaced two or three times until the active oozing is checked. If this does not occur, and if a definite bleeding point is seen, it is caught with one or two artery forceps, which are allowed to hang on for a half minute. While waiting for the patient to awaken we fold into the postnasal space a strip of gauze (known as a Mayo sponge) from six to eight inches long and two inches wide. Releasing the catheter which allows the soft palate to come down on this Mayo sponge, every particle of oozing is checked. When anesthetic is discontinued, and the patient begins to react, the anesthetist pulls once more on the catheters and the gauze is removed from the postnasal space. Before removing the catheters the anesthetist always wipes off their free ends with alcohol, since in pulling them out they must pass the operated field and might carry infection.

After-Care.—Great care is taken in the transfer of patient from operating room to his room to cover him thoroughly, because the body is usually wet from perspiration. The patient's bed, as after any other operation under general anesthesia, should be warm. The head of the patient should lie flat on the bed.

Sips of water may be given earlier than was formerly thought advisable, because the stomach is not filled with blood, and hence the patient does not vomit as readily. If sponges are employed they are removed early in the afternoon, if the case is operated in the morning, and on the next morning if the operation was performed in the afternoon. By depressing the tongue and cutting the black silk thread with a blunt pointed scissors the pillars will separate and in most cases the rubber sponge will roll out. If not, then it may be teased out with the same scissors.

The diet in the first twenty-four hours consists of water, tea or milk. The mouth and throat should be gently rinsed with a weak solution of permanganate of potash solution as often as the secretions accumulate. The diet should be increased every day, but usually on the third or fourth day the difficulty in swallowing is greater than at first, due to the infiltration, which is best managed by the application of tincture of iodin to the tonsillar wounds.

Under Local Anesthesia.—(a) The patient is asked to eat as usual, and under no circumstance should he submit to operation on an empty stomach, even though the patient may gag and vomit up the meal. This is not on account of the lesser toxicity from cocain, but because starvation weakens the patient somewhat. Morphin and atropin are usually given hypodermatically.

(b) The patient is put in a sitting posture. The whole nasopharynx and hypopharynx are swabbed with a solution of adrenalin 1/1,000. This is followed with the careful application to all these parts of pure flake cocain. The nasal cavities are also cocainized in the inferior meatuses, as rubber catheters may be used.

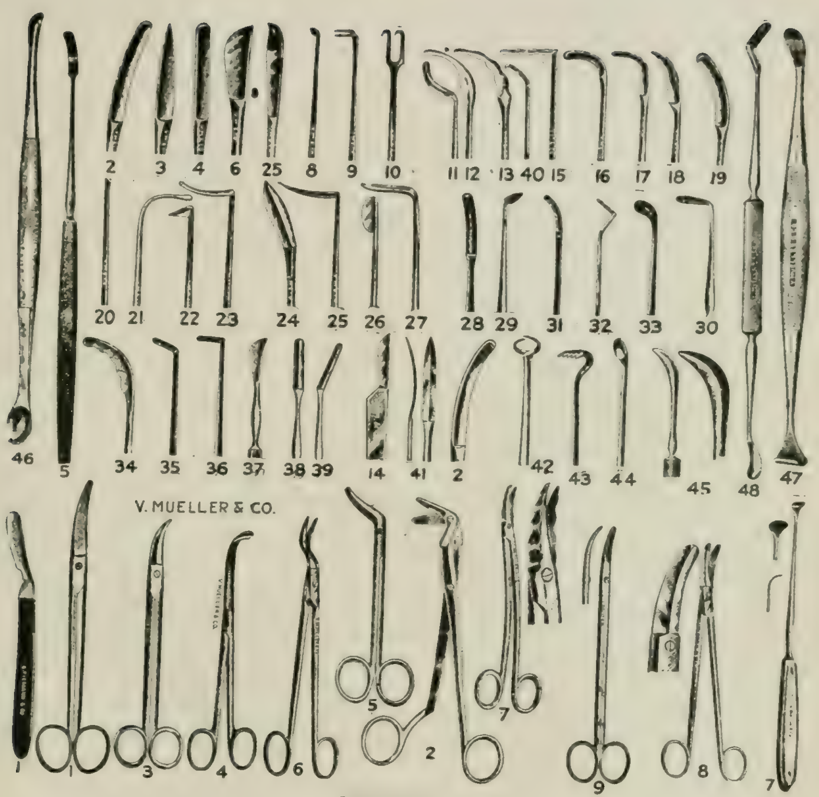
(c) The patient is now placed on a table in a semireclining position and an injection of about ten minims of a one-half per cent solution of novocain is made in the vicinity of the sphenopalatine foramen, by way of the mouth, thus further blocking the sensation. He is then given a towel to expectorate in, so as to obviate the necessity of rising and disturbing the whole field of operation. Use is made of the suction apparatus. The operative technic is the same as when under general anesthesia, except that a great deal more effort is required to raise the tonsil into the supratonsillar fossa, and the technic is more difficult on account of the nervousness and gagging of the patient. In some of these cases it is not possible to introduce the catheters until the tonsil has been removed.

Dissection Operation.—(1) The same general or local preparations of the patient are employed as in the other operations.

(2) After the catheters are passed through the nose the tonsil is grasped by means of a vulsellum forceps and drawn towards the median line. A small incision with the writer's knife (Figure 13) is made at the superior pole of the junction of the tonsils and plica supratonsillaris down on to the capsule. With the same knife this incision is extended anteriorly and posteriorly along the two pillars. By means of Mayo's curved scissors (Figure 14) the tonsil is further freed from its attachment, always putting on an artery forceps before severing the attachments near the base. When the tonsil is free on all sides it is released from the vulsellum and surrounded by the tonsillectome and grasped again by the vulsellum, then is pulled through the ring, being sure that the lower pole is included. The wire loop is now released and drawn down, severing the remaining attachments. The management of the tonsil cavity is the same as described before.

As said before, the dissection operation is performed only when we cannot dislodge the tonsil completely by means of the tonsillectome, whether under local or general anesthesia.

I have gone over many of the points in detail advisedly, although most men doing tonsil work are familiar with the required technic, the great majority of operators will adopt the technic which they can best master and by which they secure the best results.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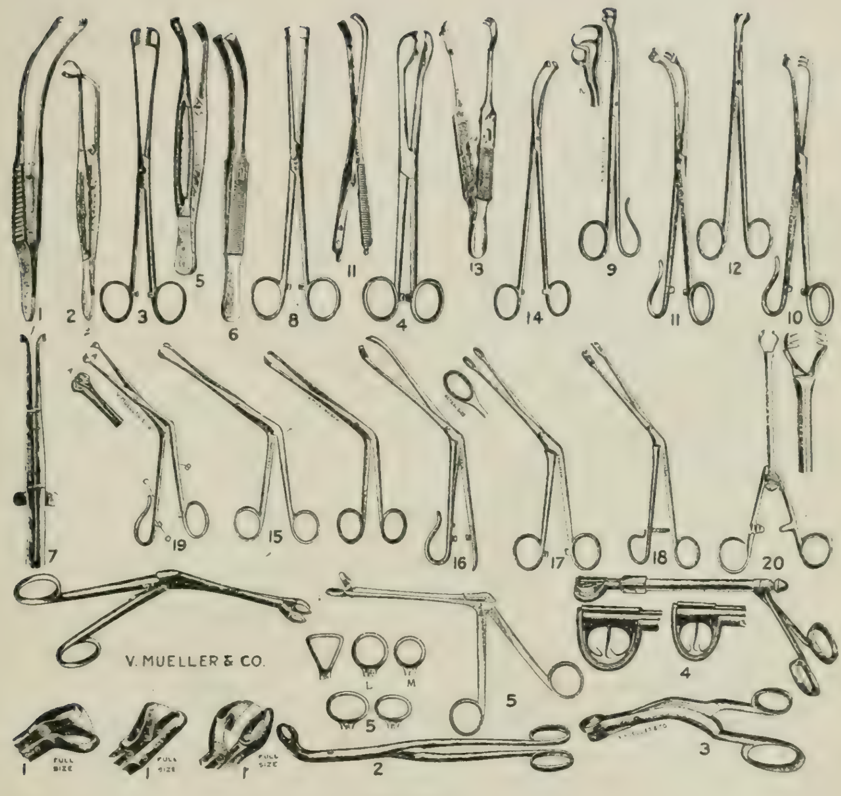
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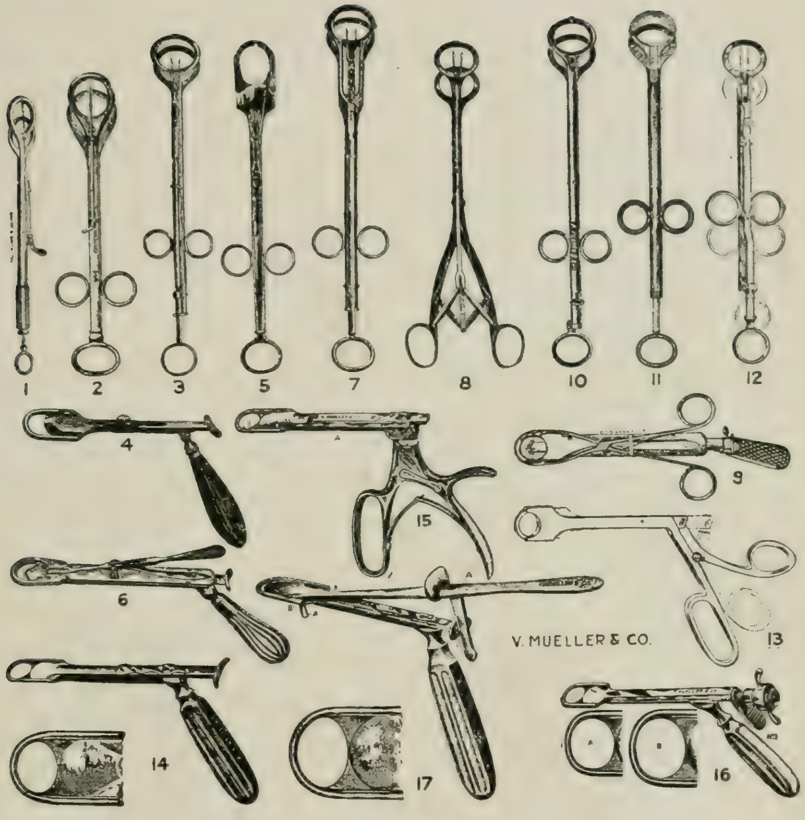
FIGURE 2.



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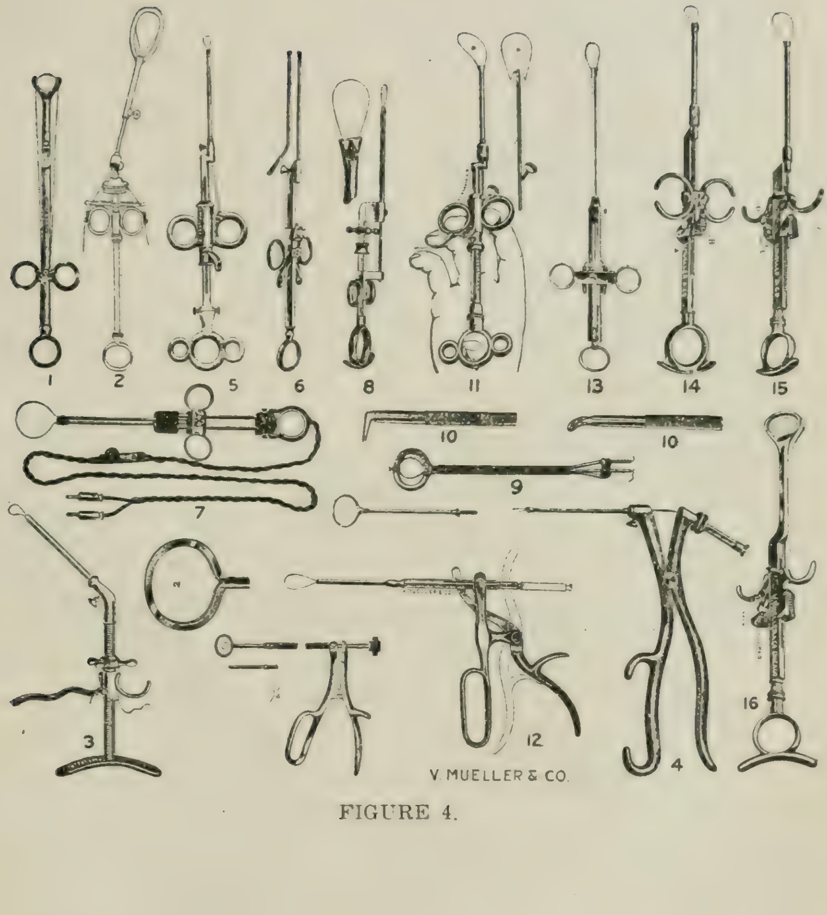

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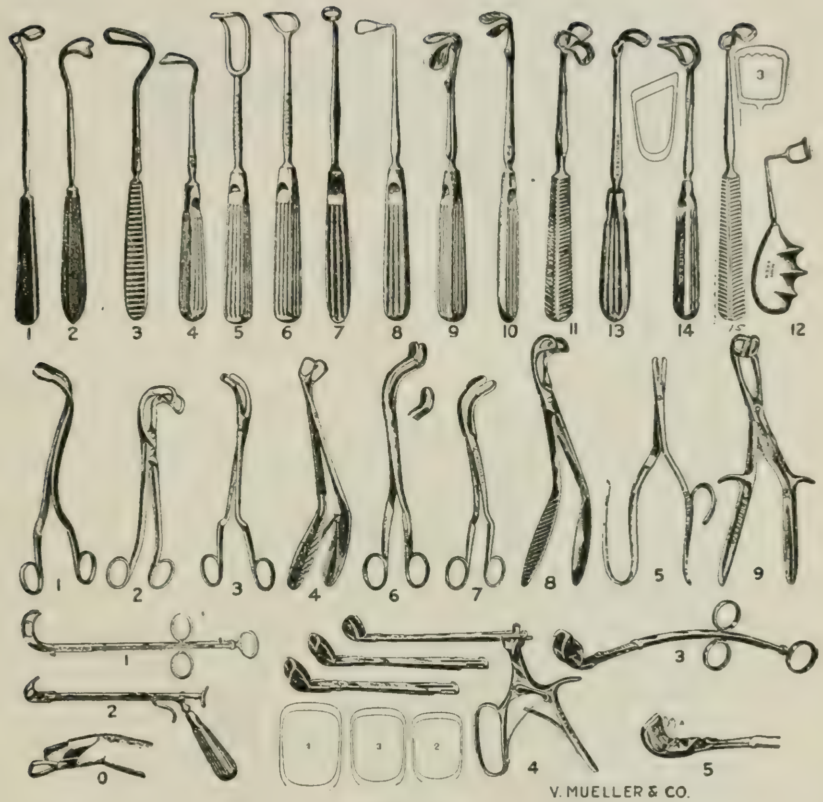
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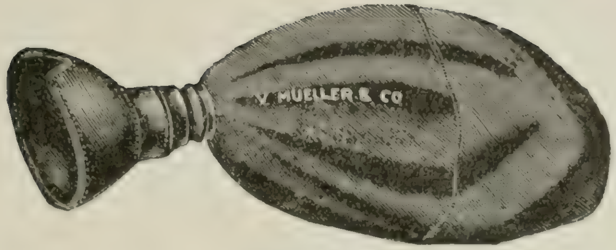

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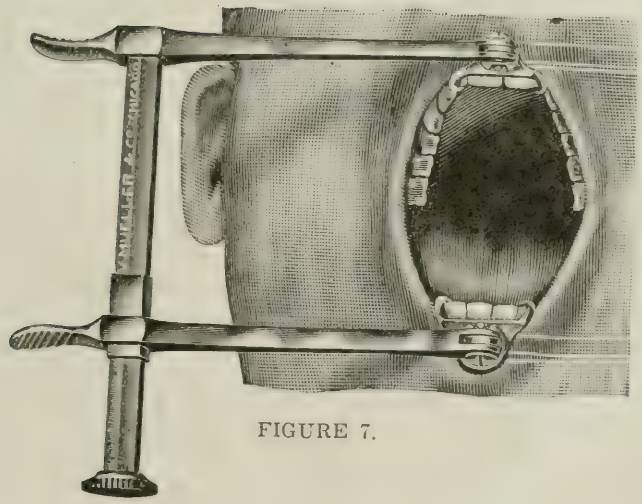

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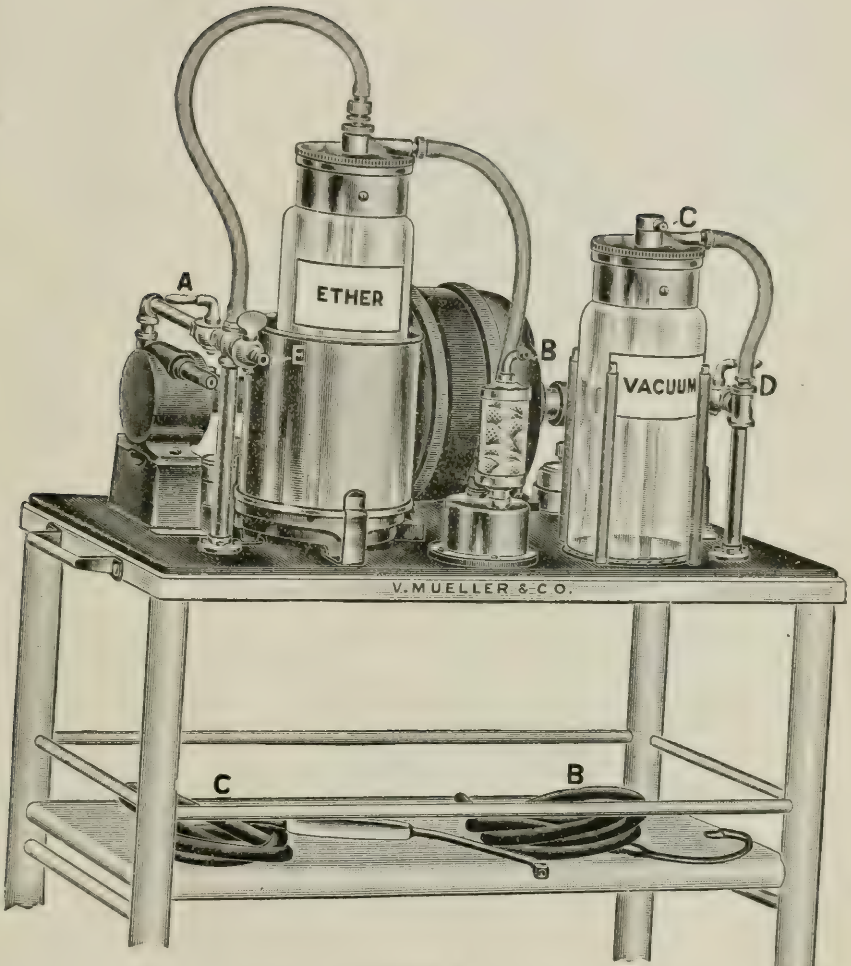

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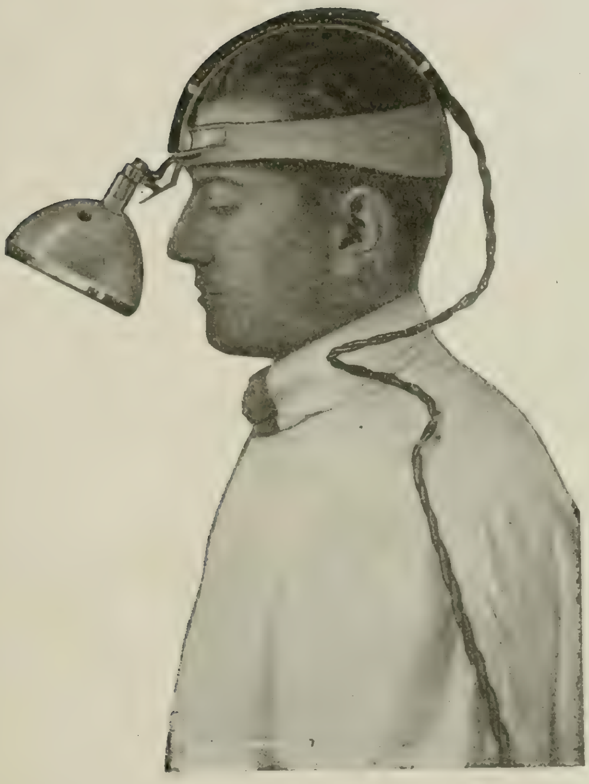

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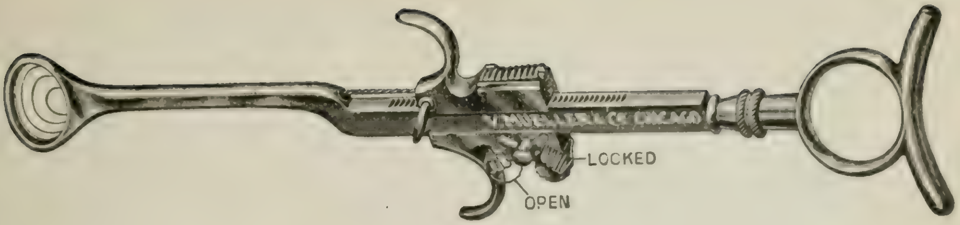

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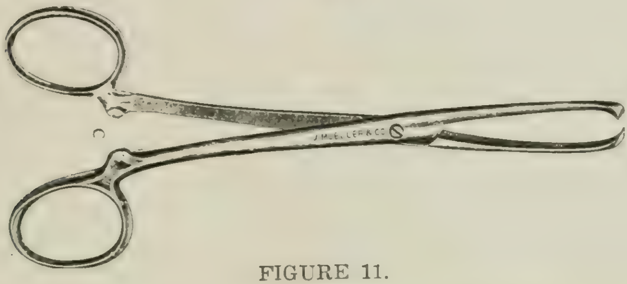
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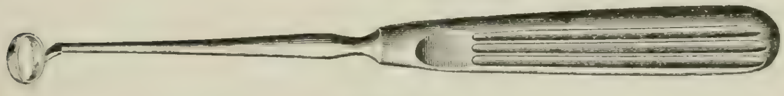

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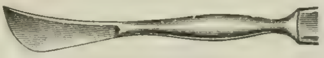

FIGURE 13.

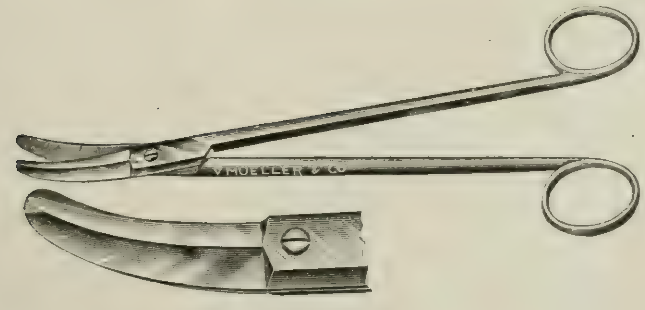
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FIGURE 14.

XV.

DIAGNOSIS OF INFLAMMATORY DISEASES OF THE LABYRINTH.*

By John B. Rae, M. B., C. M.,

New York City.

Any discussion of the symptomatology of inflammatory conditions of the labyrinth should now call for some definite method of classification. There are two such conditions whose identity is well established, and they may very well serve as a starting point from which other conditions may be approached whose exact classification may still be considered as open to debate.

The first of these is acute diffuse labyrinthitis. This is one of the conditions which was responsible, and rightly so, for the introduction of the qualifying term "manifest." A "manifest" labyrinthitis is one in which the patient exhibits spontaneously symptoms which can directly be referred to the labyrinth.

The second is the dead labyrinth, the result of chronic diffuse suppuration either still active or residual, and is the type to which the term "latent" has been applied in contrast to the "manifest" type. In the latent type the hearing is quite lost and the vestibular function is completely ablated. The same clinical picture will be presented when the patient under examination has been subjected to the complete labyrinth operation.

The chronic diffuse or latent type is the terminal stage of the acute diffuse type, and the progress from the one to the other may be extremely rapid. Acute diffuse suppuration of the labyrinth will completely and permanently destroy the end organs of both the cochlear and vestibular branches, so that within a few days, or at most a few weeks, the two conditions can only be clinically differentiated by the reaction to turning and by the galvanic tests.

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*Read at the annual meeting of the Medical Society of the State of New York at Saratoga Springs, May 16, 1916.

When we come to consider the socalled "circumscribed" cases, we enter upon debatable ground. It is impossible to conceive that any active suppurative process beginning, for example, at a break in the labyrinth wall in the neighborhood of the horizontal canal and really invading the endolymphatic spaces, should remain circumscribed. On the other hand, in such cases, that is those of fistula in the horizontal canal, it is a matter of common clinical observation that these patients present spontaneously at intervals, symptoms which undoubtedly are referable to the vestibular apparatus and which at first glance would bring them under the "manifest" type. But on examination we find them to possess hearing, and the vestibular function is not only maintained, but is abnormally sensitive to experimental stimulation. On the subsidence of the attack, these patients present clinically the appearance and tests of the ordinary chronic middle ear suppuration.

In another class of cases also, the subjects of chronic suppurative otitis media, but without a fistula, similar vestibular attacks occur. This similarity is very striking. In both varieties there is undoubtedly at the time of such attacks a disturbance of pressure within the labyrinth. This may be vascular in character, amounting perhaps to serous effusion, and due to local changes outside of the labyrinth. On restoration of normal pressure conditions the status of the patient is exactly as it was before the attack. Both of these types have one feature in common. They are both the subjects of chronic middle ear suppuration, often of long duration, and are both very liable to have bony changes in the antrum, mastoid cells and aditus ad antrum. They differ only in that in the fistula case, such bony destruction can be demonstrated; while in the other, such a condition is not capable of clinical proof.

The writer, in accordance with the views briefly mentioned, ventures to suggest that we would be advancing the cause of more exact classification if in the meantime we entirely omitted the use of the term "circumscribed" labyrinthitis, and if in its place we described these cases as peri- or paralabyrinthitis with or without fistula.

It will be observed that the further qualifying terms "serous" and "suppurating," as applied to labyrinthitis, have so far not been mentioned. In the very earliest stages these forms cannot be differentiated. Indeed, the serous variety is very likely the first stage of the purulent. The general statement may be made that the case which progresses rapidly to destruction of both hearing and vestibular functions is a purulent case, whilst that which runs a less acute course and terminates in resolution with more or less restoration of function has been of the serous type. In other words, the diagnosis is made on the results and not on the evidence.

This brief allusion to classification will lead us to consider the symptomatology of the following types: (1) Acute diffuse labyrinthitis, (2) chronic diffuse labyrinthitis, (3) peri- or paralabyrinthitis with fistula, and (4) peri- or paralabyrinthitis without demonstrable fistula.

(1) Acute diffuse labyrinthitis.

Acute diffuse labyrinthitis occurs in the course of acute or chronic otitis media. In the former case infection is most likely metastatic, with a resulting empyema of the labyrinth. In the latter case the labyrinth is most commonly exposed to infection by extension of bony caries. In both cases the course is similar. The onset is sudden and the urgent symptoms are referable to the vestibular apparatus, namely, vertigo with nausea and vomiting, disturbances of equilibrium and spontaneous nystagmus. The vertigo is intense, so that any movement of the patient may determine a violent attack of vomiting, and the disturbance of equilibrium is such that he is unable to walk. The nystagmus is most commonly rotatory in type, is to the affected side, and is to be observed in all positions of the eyes, most markedly, however, an abduction in the direction of the quick component.

Should the patient be made to stand erect, it can be observed that he will tend to fall in a direction opposite to that of the quick component of the nystagmus, and that the direction of falling can be changed by changing the position of the head.

It is obvious, however, that the patient is in no condition to be removed from bed, and the writer wishes to reiterate and emphasize the statement he made in a paper read before the American Otological Society in 1912, namely, that the spontaneous evidence in these acute cases is so informing and convincing that we are in no need of experimental testimony in addition. Accordingly, such patients need never be subjected to caloric and rotation tests. In plain English, during the acute attack, so long as the spontaneous nystagmus is to the diseased side, the vestibular apparatus on that side is functionating and no further information can be gained by subjecting this sick patient either to the caloric or rotation tests.

Should nystagmus to the bad side cease and nystagmus to the good side begin, this in itself is presumptive evidence of the destruction of the diseased end organ, especially when associated with loss of hearing. Caloric tests can now be made without much added discomfort to the patient, but will give no further information of real value.

The symptoms so far have only referred to the vestibular apparatus, and on this evidence alone the case could not be called a "diffuse" one. The functional examination of the hearing is of absolute importance. In the earliest stages the result of the hearing test is determined by the middle ear condition. The patient is deaf for the low notes, hears the high notes, has increased bone conduction, Rinné is negative, and the Weber is referred to the bad ear. With destruction of the end organ of the cochlear branch total deafness ensues. This must be determined when the hearing ear has been carefully excluded by means of a suitable noise apparatus. The hearing tests should be repeated at short intervals, because it is only when we discover total loss of hearing in conjunction with a complete lack of vestibular function, that we are able to make the diagnosis of a "diffuse" labyrinthitis.

With the destruction of the end organ of the vestibular nerve, nystagmus to the sound side is to be observed and is to be explained by imbalance of centers by the sudden cessation of impulses from one peripheral organ.

This nystagmus is also accompanied by vertigo and disturbances of equilibrium, following the rule as described for the primary nystagmus. Should no complication arise, these secondary symptoms gradually subside, and in a longer or shorter period, when balance of centers has been reestablished, are not to be observed at all.

This is the course, in general, of acute diffuse suppurative labyrinthitis. Should the process have been serous in character without destruction of end organs, complete loss of hearing does not result at any stage, nor do the secondary vestibular phenomena attributable to the center on the good side arise. With the subsidence of the inflammatory process, hearing returns more or less to its previous condition and the vestibular appaartus is gradually restored. In process of restoration nystagmus is irregular to either the good or the bad side.

A variety of labyrinthitis has been described under the name of "acute diffuse secondary labyrinthitis," and has at least in some instances been attributed to trauma arising from the performance of the radical mastoid operation. It has been the experience of the writer, and of those of his colleagues with whom he has discussed it, to meet few if any of these cases, and as the course of this variety does not differ in any way from one or other of the forms already described, it does not seem needful to complicate the classification by its addition.

It seems proper at this point to repeat that the serous and purulent variations cannot be differentiated at the outset by any means known to the writer. The rapid progress and early destruction of the end organs on the affected side undoubtedly point to the purulent type. Lack of these would seem to indicate the serous type.

Following acute diffuse suppurative labyrinthitis there is, it is hardly necessary to note, no restoration of either cochlear or vestibular function. The case gradually passes into the second group to be described, namely:

(2) Chronic diffuse labyrinthitis.

These are all cases of chronic purulent otitis media, either active or residual, and must presumably at some time have passed through the acute stage described above. It is quite likely, however, that no such history may be obtained from the patient, and this need be no source of surprise, if we reflect that many of these chronic diffuse labyrinths are the result of a panotitis occurring in early childhood in the course of an attack of scarlet fever, measles or diphtheria.

This condition is usually discovered in the routine examination of chronic purulent otitis media cases, and the diagnosis is as a rule without difficulty. Hearing is entirely absent on the affected side, and the vestibular apparatus is not irritable on irrigation of the ear with cold or hot water. The patient does not suffer from vertigo, there is no spontaneous nystagmus, nor is there any disturbance of equilibrium.

When compensation has been fully established, the after-nystagmus on rotation may be equal in both directions. But while rotation nystagmus can practically always be obtained both to right and left, it is a matter of observation, especially in recent cases, that the after-nystagmus to the diseased side may be considerably shorter than to the sound side.

This brings us to the third class of cases, namely:

(3) Paralabyrinthitis with fistula.

These for the most part also occur in the course of chronic suppurative otitis media, especially in the experience of the writer, when the suppurative process is complicated by the presence of socalled cholesteatoma.

Erosion by caries of some part of the labyrinthine capsule takes place. This is a slow procedure, and permits of the walling off, by adhesions at the point of exposure, of the perilymphatic space. It might be described as a limited endosteitis.

The commonest location of erosion is in the horizontal canal as it lies in the aditus. In this locality the carious process may also involve the fallopian aqueduct with an added facial paralysis. This combination of positive fistula symptom accompanied by facial paralysis almost certainly locates the fistula in the horizontal canal. Indeed, with a dead labyrinth in which no fistula symptom could naturally be obtained, the writer has been able to deduce, and later prove on operation, the presence of a fistula in the horizontal canal by the onset of facial paralysis.

The next most common seat of erosion is in the neighborhood of the oval window, and the least frequent in the promontory. It is impossible, however, to determine exactly the site of the fistula in many cases before operation. The patient who is the subject of a labyrinthine fistula suffers at intervals from symptoms due to irritation of his exposed vestibular apparatus. As already stated, it is the opinion of the writer that these are by no means the expression of a true labyrinthitis, but are rather determined by alterations in intralabyrinthine pressure due to conditions outside the labyrinth.

These symptoms vary only in intensity and frequency from those which mark the onset of any diffuse inflammation of the labyrinth, and consist of vertigo, disturbances of equilibrium and spontaneous nystagmus. It is when we come to make our cochlear examination and apply our experimental vestibular tests that the differential diagnosis is made.

The hearing tests show the lesion to be confined to the conducting mechanism.

The compression and aspiration tests prove the presence of a fistula in most cases. It should not be forgotten, however, that even when we are dealing with a fistula—as may be subsequently proved on operation—this test may be negative. The defect in the canal may be so blocked off by granulation or cholesteatomatous material as to prevent any pressure changes occurring during the experiment. Should we on compression obtain some coarse nystagmic movements, a conjugate deviation of the eyes in one direction, with or without head movements, and should we also obtain on aspiration a conjugate deviation of the eyes in the opposite direction, the diagnosis of labyrinthine fistula may be made. In addition, we are now satisfied that we are dealing with a functionating vestibular apparatus, as it is very evident that were the vestibular end organ destroyed, we should not be able, even with a fistula, to convey any impulses to the vestibular nuclei. In other words, a dead labyrinth with a fistula will not give us a positive test.

After a shorter or longer period, from a few hours to a few days, these symptoms subside, and the case is not to be distinguished, unless experimentally, from the ordinary chronic suppurative middle ear.

The caloric reactions are usually very readily obtained, unless, as previously stated, the labyrinthine capsule is blocked off by granulation or cholesteatoma. The turning tests confirm the presence of irritability of the vestibular apparatus.

To sum up, in this type we have to deal with a patient suffering from chronic suppurative otitis media, with changes in the bone in the aditus, antrum or the mastoid cells, with a break through some part of the labyrinthine capsule, with a functionating vestibular apparatus, and whose hearing defect is limited to the conducting mechanism.

(4) Paralabyrinthitis without fistula.

This type is exemplified by the patient who suffers from chronic suppurative otitis media, who is subject at intervals to attacks referable to the vestibular apparatus, who is presumably suffering from bony changes affecting the aditus, antrum or mastoid cells, and whose hearing lesion is confined to the conducting mechanism. He only differs from the previous type in that we are unable to obtain a positive fistula test. That we find many of these cases without fistula has been repeatedly proved on operation.

The caloric and rotation tests also prove the irritability of the vestibular apparatus.

It will be apparent that no attempt has been made in the above brief remarks to go into the constitutional symptomatology of labyrinthine inflammatory conditions. The writer takes it for granted that his hearers are familiar with the experimental methods named, and has made this contribution solely in the hope that discussion of this important and interesting subject will result in a simplification of the classification, at this moment cumbersome and complicated.

CLASSIFICATION.

1. Acute diffuse labyrinthitis.
2. Chronic diffuse labyrinthitis.
3. Paralabyrinthitis: (a) with fistula; (b) without demonstrable fistula.

XVI.

THE TREATMENT OF LABYRINTHINE AFFECTIONS.*

By Wendell C. Phillips, M. D.,

New York City.

The treatment of the various affections of the labyrinth, and more especially that of the purulent invasions of this small space, has not, up to this time, been placed upon any permanent basis. The difficulties of differential diagnosis, and the fact that we know that many cases of purulent labyrinthitis recover spontaneously, even where a part or whole of the labyrinthine capsule becomes necrosed, lead to a wider divergence of opinion regarding the exact indications for operative interference. On the other hand, we must admit that there are cases where it is possible to save life by timely operative procedure. In the present state of our knowledge it would seem that at least a moderate degree of conservatism should rule the action of the surgeon, except in cases that already show the infection to have passed through the labyrinthine spaces into the meninges.

In the preparation of this short paper the writer has before him histories of thirty-six cases of the various types of labyrinthine invasions. Twenty-six of these cases have already been published, the types varying from the destructive lesions of parotiditis and tertiary syphilis to those of acute diffuse labyrinthitis. Out of the thirty-six cases there were fourteen operations upon the labyrinth, seven of which were for the removal of the extensive necrosis or sequestra in patients who had long since passed the acute stage of the disease. This leaves a record of seven operations for more or less acute labyrinthitis. There were five fatalities. One of the fatal cases occurred in my own practice from the rupture of a temporosphenoidal abscess and meningitis two months subsequent to the labyrinth operation. This patient might have been saved had he shown any symptoms, barring headache, of the large brain abscess from which he suffered. In two of the fatal cases no operation upon the labyrinth was performed. One of these cases was drained through the cisterna magna, and the other was rapidly fatal, and no operation was attempted. In one case, which recovered, a thrombus in the jugular bulb developed as the result of an accidental puncture while removing the necrosed promonotory. Two others had accompanying brain abscesses.

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*Read at the annual meeting of the Medical Society of the State of New York at Saratoga Springs, May 16, 1916.

Primarily, it may be stated that the indications for operation depend upon the type of labyrinthine involvement. Nonsuppurative cases resulting from parotiditis, hemorrhage or other effusions, and epidemic cerebrospinal meningitis, should never be subjected to operation. The labyrinth should not be operated upon in cases of serous labyrinthitis, and herein lies one of the difficulties with which we have to contend—namely, the differential diagnosis between certain cases of serous labyrinthitis and purulent labyrinthitis. In both, the destruction of the cochlea and the static labyrinth may be complete and permanent, and we are without positive differential data, barring our knowledge of the probable cause in each individual case. Complete destruction of function, however, is less likely in the serous cases. Whenever any doubt exists as to whether the case is purulent or serous in character, the patient should be given the benefit of the doubt and the operation delayed, pending further developments.

Regarding the indications for the labyrinth operation in acute diffuse labyrinthitis there is bound to be a wide variance in opinion. Its development in chronic suppurative cases seems to be more gradual, and these cases offer more hope of becoming circumscribed, and hence they are less likely to develop meningeal complications. The seven cases of necrosis or sequestra above reported seem to be confirmatory of this opinion, for they were all cases of chronic suppurative otitis media. On the other hand, it is well known that in acute diffuse purulent labyrinthitis accompanying acute purulent otitis media the infection usually extends rapidly to the meninges, and in these cases early surgical measures seem to be justified. Even though meningeal symptoms have already appeared, a complete labyrinth operation should be done, together with the establishment of drainage of the meninges at, or near, the internal auditory canal. This operation, together with the drainage of the meninges, has proven effective in a limited proportion of cases of labyrinthine involvement with meningeal complications. It would seem that the recoveries are due to the more or less localized character of the meningitis, hence the meningeal drainage would seem to be a more important factor than the operation upon the labyrinth.

I am indebted to Dr. C. J. Imperatori for the following history of recovery from an undoubted meningeal involvement as a result of purulent labyrinthitis. This case, however, occurred in a patient who had suffered from chronic purulent otitis media for many years.

Miss M. P., aged thirty-nine years.

Past History.—Had mostly all diseases of childhood. Measles when twelve years old, which left her with an otitis media purulenta chronica left; otherwise her past history is negative.

Family History.—In so far as present condition is concerned is negative.

Present History.—Three weeks ago began to have severe headaches, mostly frontal and occipital, and noticed as her headaches increased in severity the amount of urine passed lessened, so that on May 4, 1915, when she entered the Park Hospital, she was passing but a few ounces of urine. She was entered in the medical ward, and tentatively considered a case of beginning uremia. At this time she was in a more or less stuporous condition, refusing to answer questions regarding her past history, and always rambling off on some irrelevant subject. She had some rigidity at the back of the neck, and lumbar puncture was done, and because of the variety of bacteria within the spinal fluid the pathologist remarked that the fluid seemed to come from one suffering from otitic meningitis. At this, one of the internes said he remembered she had a foul smelling discharge from her ear. On May 6th, Dr. Imperatori was asked to see her. Examination[1] showed the patient lying in a stupor, from which she could be aroused and would answer questions fairly intelligently. There was decided cervical opisthotonos, but no Kernig, no Babinski, no Gordon nor Oppenheim. The other reflexes were decidedly suppressed, and there was a rotatory nystagmus to the right. She was tender over the left mastoid, and she had a very offensive discharge from the exetrnal auditory canal. There was some granulations at the bottom of the canal, but no drum or ossicles could be made out. Functional testing of the right side showed an active labyrinth. Caloric reaction within twenty seconds with water at 65 degrees Fahrenheit. Syringing the left side with water at 50 degrees produced no reaction within eight minutes. With a noise apparatus in the right ear there was no response to the voice, tuning forks or whistle. With a 256c tuning fork the patient lateralized to the right side—that is, her sound side. The eye grounds showed some paleness of the nerve vessels, with a slight atrophy of the nerve. Motility of the eyes was normal. The examination of the spinal fluid showed two hundred and fifty cells, and the variety of the bacteria being so numerous that the pathologist could not make a report from a smear diagnosis. Culturely he was able to recover streptococcus from the fluid. Operation was advised, but the family refused permission. On May 9, 1915, the patient was unconscious and could not be aroused and had all signs of terminal meningitis. Finally, the family and also the doctors gave permission: "Do your old operation, she is going to die any way," is what they said.

Operation.—Preliminary lumbar puncture showed a moderately cloudy fluid (three hundred cells) with a pressure of twenty degrees Strauss instrument.. The usual radical operation without cutting a flap was done, and this was immediately followed by a Jansen-Neumann operation upon the labyrinth. The dura was incised at the internal auditory orifice. The sinus was situated very far forward, and made the latter phase of the operation somewhat difficult. The patient was returned to the ward, and by the next morning, May 10th, was semiconscious at intervals, and by the 18th of May was conscious and rational. A lumbar puncture was done twice on May 10th removing twenty-five cubic centimeters of fluid each time. On May 11th, thirty cubic centimeters of fluid was removed; on May 12th, thirty cubic centimeters, and on May 13th, thirty cubic centimeters. Pressure of the spinal fluid varied between sixteen and thirty degrees, the cloudiness gradually disappearing from the fluid. The cerebrospinal fluid drained considerably through the mastoid wound for the first few days. By the 3d of June she was in very good shape, having regained full control of her sphincters. The quantity of urine gradually increased until the usual normal amount was passed. A plastic closure was done, and by the 1st of October there was complete epidermization of the radical cavity. The patient was able to walk alone by the 15th of July, using a cane, but there was a slight staggering gait. This completely disappeared. She has a facial paralysis on the operated side. The writer has known of another similar case.

It is not to be expected that recovery will take place in the large proportion of cases, but the fact that the meningeal infection may occasionally be more or less localized offers sufficient encouragement to warrant this attempt to save life. In these desperate cases no half-way measures are permissible, and the Neumann operation, wherein the bone is entirely removed, including the border of the internal auditory canal, together with drainage of the cerebellar spaces, is the ideal procedure.

In two cases out of the thirty-six the labyrinth symptoms resulted from traumatism at the hands of inexperienced operators while incising the drum membrane. Dr. J. H. Güntzer has reported one of these cases: On May 31, 1915, was called by the family physician to see M. K., male, thirty-one years old. Found the patient in bed lying on his left side. History of pain in right ear for two days; no discharge. Since the previous evening was unable to walk, felt dizzy and vomited. Examination of the right ear showed the membrana tympani slightly reddened, no bulging and a pinhead perforation about the center of the superior posterior quadrant with a droplet of serous red discharge exuding. The patient had a spontaneous horizontal nystagmus to the left and rotary on looking up; on attempting to rise, fell toward the right. Hearing was not impaired, no mastoid tenderness, no temperature, pulse 88. Diagnosis of labyrinthine irritation was made, but no assignable cause could be elicited. Calomel and sodium bromid were recommended, rather as placebo. This condition lasted ten days before the patient was able to leave the bed. At a subsequent office visit the patient confided that his family physician was called the evening before my first visit to the patient. The patient was told he had an abscess in his ear, and the doctor used an instrument to open it, when the patient at once became dizzy, nauseated, vomited, and was unable to walk.

The labyrinthine irritation was most likely due to disturbance of the foot of the stapes. Fortunately, the middle ear and labyrinth were not infected, there was no discharge, and the patient escaped the dire consequences that may have followed such an infection.

In the second case both hearing and vestibular functions were completely destroyed. Neither seemed to be of the purulent type, and it is probable that sufficient injury to the stapes was produced to cause either hemorrhage or serous exudate in the labyrinthine cavity.

It should be noted that in certain cases of chronic purulent otitis media, the patient complains of recurring attacks of vertigo. Here it will often be found, upon investigation, that such attacks occur just previous to the throwing off of large masses of cholesteatomatous material. These socalled paralabyrinthitis cases should invariably be subjected to the radical mastoid operation, in order to prevent further erosion of the labyrinthine capsule. In fact, in nearly all patients suffering from chronic purulent otitis media who have vertiginous attacks, without signs of involvement either of the auditory or static labyrinth, the radical mastoid operation should be done. This procedure is followed usually by a cessation of the symptoms. In the type known as the circumscribed labyrinthitis, where there are labyrinth symptoms with a normal rotation test, a positive caloric reaction and a positive fistulæ test, the radical mastoid operation should be done, but the intact membranous labyrinth should not be disturbed. In cases where the labyrinthine capsule is found to be more or less necrosed and sloughing, or where sequestra involving any portion of the labyrinth are found, the necrotic areas should be removed, but, so far as possible, the operator should avoid extending the procedure beyond the lines of demarcation; in other words, he should not go beyond the walled-off areas. Here a partial labyrinthine excavation is justifiable.

Finally, a word of caution, in performing the radical mastoid operation, is here ventured. It is a fact that in a considerable proportion of cases of fatal labyrinthitis reported the histories state that the labyrinth symptoms appeared soon after the performance of the radical mastoid operation. It is fair to assume that in many of these cases the labyrinth invasion results from careless operating, wherein the stapes is dislodged from its position in the pelvis of the oval window, the round window is entered, or the bony capsule is punctured by chisel or curette. These accidents are unjustifiable and should not occur.

This paper has been written with the intent of provoking discussion and bringing to light the opinions and experiences of other observers, in the hope that we may more rapidly reach some definite basis regarding the indications for surgical measures.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of October 13, 1916.

Atypical Sinus Thrombosis.

Dr. Hugh Blackwell: The patient was first seen on April 15, 1916. At that time he gave a history of having had a discharging ear for two months. For the last two weeks he had suffered from severe headaches—chiefly nocturnal—and some nausea.

Aural examination revealed a marked sagging of the posterior wall, profuse discharge, very acute general mastoid tenderness, and marked mastoid deafness.

The patient was immediately sent to the hospital, and was operated upon the same day. On opening the mastoid it was found to be filled with pus. Smears from the cells showed the presence of streptococci. The sinus plate was found to be diseased and was partially removed, the sinus being exposed for a considerable area in its vertical portion. It was of normal appearance. For the following eleven days the patient's convalescence was uneventful. On the twelfth day of convalescence, without any apparent chill, the patient suddenly developed a temperature of 105.4°. During the following nine days there were seven distinct rises of temperature. Five of these rises reached points btween 104.4° and 105.4°, and two reached 103.6°. These accessions of temperature were followed by five intermissions to normal and by two marked remissions. Throughout this period the patient was under the most careful observation, in order to detect any evidence of chills or chilly sensations. No trace of this symptom was present; the patient did not even complain of being cold prior to the rises of temperature. The remissions were not accompanied by sweats.

Immediately after the first rise in temperature a blood culture was taken, which proved to be negative. Blood count showed 11,000 white cells present, of which eighty-nine per cent were polymorphonuclear leucocytes. The mastoid wound was negative in appearance, as was also the exposed sinus. The middle ear had ceased to discharge.

Eight days after the initial rise of temperature a second blood culture was taken, which showed numerous colonies of streptococci, the blood count showing 13,000 white cells and a lowering of the polymorphonuclear count to seventy-nine per cent. In the meantime some chest râles developed, and slight tenderness at McBurney's point. Dr. Wyckoff, our internist, made several examinations of the chest and abdomen, and finally decided that there was nothing in these cavities that could account for the temperature.

On the ninth day of temperature the patient was operated upon a second time. A well organized clot was found, completely occluding the torcular end of the sinus. The clot was removed and the jugular vein was excised. Subsequently the patient's temperature dropped and he made an uneventful recovery.

The only atypical feature worth noting in this case was the absence of anything remotely resembling a chill or sweat. Dr. Blackwell, of course, was aware that the absence of chills in sinus thrombosis has been noted before, especially in children; but it is rare to see so many rises of temperature as this case presented—seven in all—associated with a septicemia, without any chill. The case illustrates the value of the services of a skilled internist in excluding chest and abdominal complications as a cause for the temperature. The blood culture was purely a confirmatory aid in reaching the diagnosis. The absence of a bacteremia was to be expected in the initial culture, as was the presence of a positive culture after the condition of septicemia became established.

Paper: Septic Sinus Thrombosis of the Jugular Bulb, With Repeated Formation of Thrombi in the Sigmoid and Lateral Sinuses—With Special Reference to the Literature of the Involvement of the Torcular Herophili in Such Cases.*

By John Randolph Page, M. D.

DISCUSSION.

Dr. John D. Richards said that he had had the pleasure of seeing this patient with Dr. Page, and that aside from the anatomic features of the torcular, brought out in Dr. Page's paper, there were certain pathologic facts which may in specific instances give an idea of the extent to which a thrombosis is likely to invade a vessel, and as to whether it will or will not pass to or beyond the torcular. The point which he wished to bring out is especially well illustrated in the jugular vein, as we study the conditions in that vessel, in cases of jugular resection for sinus thrombosis.

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*See page 595, September, 1916, number.

First, we find frequently in cases of sigmoid sinus thrombosis, when the clot has blocked the vertical portion of the sinus and also the bulb, that the upper portion of the jugular is either occluded by a clot or else is collapsed; the block or the collapse in the jugular, however, extends only to the facial vein, but not below it. The lower portion of the jugular in these cases is well filled, and circulation is taking place between the facial and the jugular. If we are careful to note in this type of case the condition of the vein wall in the collapsed or thrombosed portion above the facial, we will find that in the vast majority of cases the vessel wall shows no visible phlebitis. In such cases the thrombus may be fairly extensive, but the phlebitis remains local; and while the thrombus may extend far beyond the limits of the phlebitic area, yet it stops short at the first great tributary or emissary, and upon the same principle and for the same reasons as does the clotting in veins following the ordinary process of ligature.

Second, in the other class of cases where we find upon opening the neck that the jugular vein is collapsed or thrombosed below the facial as well as above that point, we invariably find that the jugular vein wall is involved in a phlebitis. The same thing applies to the sigmoid sinus. A phlebitis is no respecter of either tributaries or emissaries; a thrombus is. When, therefore, we find a sigmoid sinus which as we progress backward toward the torcular by successive operations becomes more free of the evidence of phlebitis in the outer wall, we may with fair confidence assume that the contained thrombus in the vessel, while it may extend back to the torcular, will limit itself there.

Dr. E. B. Dench said that in a general way he agreed with Dr. Page, and that one should always continue until the sinus wall appears healthy. A healthy sinus wall, however, cannot always be determined, for although the sinus may look healthy macroscopically, infiltration of the sinus wall with pathogenic bacteria may be present. Dr. Dench said that he had frequently incised the internal jugular where the macroscopic appearance of the vein was normal and where the vein contained no clot, and that a microscopic examination of the wall of the vein showed infiltration with streptococci. In other words, the extension of the infiltration of the wall of the vein is more rapid than the formation of the clot, and it is therefore necessary to go well beyond the clot in order to get well beyond the infiltration of the sinus wall.

Dr. Dench also considered that the anatomic part of Dr. Page's paper was valuable. He did not think that it made so much difference whether the lateral sinus of one side was continuous with the lateral sinus of the other, or whether the lateral sinus of one side was continuous with the opposite superior longitudinal sinus. The important point was that the sinuses entered the torcular irregularly, and consequently pressure applied to the torcular would probably seal the unopened sinuses running into this reservoir. The shutting off of the torcular would take place by the adhesion of the walls of the various sinuses. Such an occlusion would be aseptic in nature. Dr. Dench said that he had lost one case because he had not exposed the sinus far enough toward the torcular.

Dr. J. R. Page, in closing, said that he agreed with Dr. Richards about the phlebitis and the progress of those cases; but even where there was involvement of the vein wall, he thought it would rarely extend to the opposite side, for the reason that the main channel did not lead to the opposite side. In a case reported by Liddell, with a right lateral sinus involvement, two-thirds of the sagittal sinus was involved before the phlebitis extended across to the opposite lateral for a short distance. The tendency is for the clot to proceed to the sinus of which it is a continuation. It does not, as a rule, pass beyond the communicating branch. In the case of Simpson, the clot extended into the straight sinus without crossing to the other side. The patients usually die before it extends to the other sinus.

Paper: Report of a Case of Tubercular Mastoiditis—Radical Operation Under Cocain Anesthesia.*

By Harold Hays, M. D.

DISCUSSION.

Dr. Thos. J. Harris said that Dr. Hays' report of this case, with suggestions along the line of local anesthesia, was timely, for it illustrated what few of us possibly remember, that the subject has been thoroughly studied by Dr. Day of Pittsburg, who had treated of all the points presented by Dr. Hays.

Dr. Dench said that it was a question whether a patient who received five-eighths of a grain of morphin in five hours was having local anesthesia simply.

Dr. Harris, responding to Dr. Dench, said that Dr. Day had not used morphin. The French also had studied the subject and had done a great deal of such work under local anesthesia.

Dr. Guttman asked how much time was occupied by the operation.

Dr. Hays replied that the time was about an hour.

Dr. Guttman said that he had performed a radical operation about a year ago under local anesthesia, using hyoscin and scopolamin instead of morphin, and then infiltration with novocain and adrenalin, and that his experience was similar to that of Dr. Hays. It required two hours to perform the operation. In operating he had had no trouble until the middle ear was reached. The patient had no sensation of pain and did not complain, but the middle ear was very sensitive; so he had not tried the method again. He had also used the so-called Neumann method for anesthetizing the middle ear, and notwithstanding all this, the pain was very acute. Otherwise, the operation was very satisfactory. The field of operation was absolutely bloodless on account of the adrenalin.

Dr. Dench said that he had not understood how strong a solution of adrenalin had been employed.

Dr. Hays said that he used a 1/1000 solution.

Dr. Dench said that he had used a solution of this strength several years ago for the control of hemorrhage, and a good deal of sloughing had resulted.

Dr. Haskin said that he had operated on several cases under local anesthesia, and that it was not necessary to employ morphin. One patient upon whom he had operated was suffering from Bright's disease, and morphin was contraindicated. This patient had no pain and made an excellent recovery. Two other cases were operated upon last winter, with no trouble whatever. He always injected the canal of the ear as well as the tissues over the mastoid. When doing a mastoid under local anesthesia, the results depend not so much on the anesthetic employed as upon the method of operation. If a drill or electric burr is employed, the patient is spared much of the fear and dread occasioned by the chisel. One of these patients talked all the time during the operation. Dr. Haskin said that he did not understand why the burr is not more frequently employed; it is the easiest and safest way of taking down the canal wall and removing the outer cortex. One has only to be a little careful with the manipulation, and it leaves a clean beautiful wound, without the necessity for any scraping. In his hands it had proved much more satisfactory than the chisel and curette.

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*See page 109.

Dr. Herzig stated that he used urea and quinin hydrochlorid four per cent solution, as a local anesthetic in two cases of mastoiditis, where a general anesthetic could not be given, instead of cocain. He injected the solution in and around the region of the primary incision, and also into the upper wall of the auditory canal, just as one does to do an ossiculectomy. He experienced no difficulties in operating, and the patient suffered no pain, even when the antrum was reached. The time elapsing from the time of injection and operation was thirty minutes in each case. No toxic symptoms were present, and no preliminary injections of morphin were given. Both of the cases were diabetic. There was no sloughing aside from that normally expected in such type of cases.

Dr. Hays said that he had no intention of reporting this case as an original method of operating, since he was perfectly aware that it had been done before; but that sometimes it is well to impress the possibilities of a method that is not frequently employed. Nearly everyone has patients suffering from diabetes or nephritis where the method may be advantageously applied. Although he knew that the operation had been done by others, yet he went about it rather fearfully.

It did not make very much difference whether the loss of sensation was produced by the morphin or the cocain, provided the anesthesia was secured. Sometimes it is not desirable to use general anesthesia, and in such cases he would not hesitate to use local anesthesia, as in this instance.

Adenocarcinoma of the Hypophysis Cerebri Complicating a Chronic Purulent Otitis Media.

Dr. Wesley C. Bowers presented a preliminary report of this unusual case, which will be reported in full later.

DISCUSSION.

Dr. Dench told of performing a similar operation at the New York Eye and Ear Infirmary, doing it in two stages. Twenty-four hours after the exposure of the dura, the patient went into a collapse. He then opened the dura and found the auditory nerve trunk involved and the dura. A microscopic examination of the tumor showed it to be an adenomatous growth of the pituitary body. It was another case of those very rare growths which extend along the entire surface of the base of the skull.

Paper: End Results in the Radical Mastoid Operation.

By Thomas J. Harris, M. D.

DISCUSSION.

Dr. Dench said that if he were to open and close the discussion in three words, he would say "that he disagreed." The operation under consideration has, as the writer of this paper stated, been employed for twenty years, and yet the writer of the paper presents only eighty-five cases—and these not his own—upon which to decide the merits of the procedure. Dr. Dench said that his own operating book showed that over seven hundred cases had been performed in his own clinic and private practice, either by himself or by his assistants. Certainly, if he had been able to show only fifty per cent of cures in these cases, he would be ashamed to stand before the section and discuss the operation. He agreed with Dr. Harris that there are three factors to be considered: the cure of the suppuration, the danger to the life of the patient, and the preservation of the function of the ear. The question of danger to life, he considered to be absolutely nil. In the seven hundred and thirty-four cases operated upon, either by himself or his hospital assistants, not a single death could be directly attributed to the operation. In the paper read on this subject before the International Otological Congress at Bordeaux, he reported about one hundred cases in which the operation had been performed. One of these cases died of meningitis, but this meningitis was probably present before the operation. At that time we did not have the elaborate methods for determining the presence of meningitis which exist at the present day. So far as danger to life is concerned, with a properly conducted anesthesia the operation is as free from danger as is an interval operation for appendicitis.

In regard to the hearing, Dr. Dench said that in nearly every article he had written he had stated that if the patient had poor hearing, with no nerve lesion, the hearing would probably be improved by the operation. If the hearing in the affected ear was only moderately impaired, then the hearing would probably be the same after operation as before operation. If the hearing in the affected ear was very good before operation, it would probably be somewhat impaired by the radical procedure. In other words, if the hearing is very poor, and this impairment is due to a middle ear lesion alone, the hearing will probably be improved by the radical operation.

With reference to Dr. Harris quoting Dr. Dench as saying that in all cases of cure the discharge had not entirely ceased, this statement was absolutely wrong. The cases where slight discharge still persisted after operation were not reported as cured, but improved. The percentage of cures, therefore, mentioned in Dr. Dench's article quoted by Dr. Harris was absolutely correct. Dr. Harris stated that in some of the cases which he had examined there was profuse discharge after several months. Dr. Dench said that in cases where the discharge persisted for months after the operation, he would certainly operate a second time. In private cases, all discharge ordinarily disappeared in from two to four weeks. In hospital cases, the period of convalescence was somewhat longer, owing to the fact that certain minute details in technic were apt to be overlooked in the dressing of hospital cases—where a large number of cases have to be attended to in a given space of time. In his private work, almost invariably the patient could be sent out of the city at the end of two weeks, or two weeks and a half, with perfect safety; and if the ear was not completely dry, the subsequent after-treatment was so simple that it could be carried on either by the patient or by any general practitioner.

Dr. Harris had said nothing as to whether these cases were allowed to dermatize, or whether a primary or secondary skin grafting had been used in the cases reported. The use of the primary skin graft certainly shortens the period of convalescence, and should be used in all cases with very rare exceptions.

With reference to operating upon cases where both ears were involved, Dr. Dench made the following rule: If both ears were the seat of suppuration, and both demanded operation, Dr. Dench operated upon the poorer ear first. If after the discharge ceased the hearing in the ear operated upon was much improved, and was good enough for the patient to follow his vocation, the opposite ear was then operated upon. In the case of total deafness upon one side, and a suppurating otitis upon the opposite side, Dr. Dench said he would naturally hesitate to operate upon the ear on which the patient depended entirely for his hearing. He would not operate upon such a case unless the symptoms were very severe. In spite of all that has been said regarding the danger to the hearing of the radical operation, Dr. Dench spoke of several cases in which both ears had been operated upon, and in which the hearing was improved by the operation—in fact, one of the cases, after the operation had been performed upon the poorer ear, came back and requested that the other ear be operated upon because the operation upon the first ear had so greatly improved the hearing.

Dr. Dench said that the more of these operations he performed the better he performed them. The operation is a difficult one, and the technic of the operator influences greatly the result. With proper technic, good results can be secured, and the man who has performed the most operations would probably have the best results. He objected to the fact that anyone should take eighty-five cases of this character at random and put them forward as the end results of an operation which had had a life of twenty years. Now, as to the selection of cases, Dr. Harris had stated that by means of the X-ray plates, and by physical examination of the ear, one can form an accurate opinion as to the extent and nature of the involvement. Dr. Dench absolutely disagreed with this, and cited a case recently operated upon in which the physical examination of the ear would lead one to think that there was very little intratympanic involvement. At the time of operation the dura was found exposed over an area about an inch square. The X-ray pictures in these cases will not show you the location or extent of intratympanic involvement.

Now regarding the eustachian tube, everyone knows that a middle ear discharge confined to the eustachian tube is not dangerous. While, of course, one would prefer to secure a perfectly dry ear as the result of operation, a mucous discharge from the eustachian tube will never lead to any intracranial involvement.

Dr. Dench said he wished to impress upon the men present that the radical operation was not a dangerous procedure, and only to be used in extreme cases, but that it was a procedure which should be used in the majority of cases of chronic suppuration. It was impossible to say by looking at an ear just which cases were dangerous. The mere fact that there is a large opening in the drum membrane does not render the case safe. The drainage from the upper part of the tympanum may suddenly be shut off, in spite of a large opening in the drum membrane, and then intracranial complications may follow.

Dr. Yankauer said that he was very glad, indeed, to have heard Dr. Harris' paper, for it gave him an opportunity to compare statistics from the closure of the eustachian tube through the intact external canal, an operation which he had described some time since.

According to Dr. Harris, barely fifty per cent of cases operated upon radically result in dry ears. Dr. Yankauer said that his own statistics showed fifty-two per cent of cures by closing the tube.

In regard to the results of hearing, decided difference obtains. The experience of all has shown that the hearing is lost in most instances after the radical operation, and Dr. Harris' report confirmed that. In the cases collected by Dr. Yankauer last year, fifty-two per cent were cured, and in forty per cent the hearing was reported improved, making ninety per cent improved hearing after operation. The same is true of cases of tinnitus. Ninety per cent showed improvement in the tinnitus.

The radical operation is an operation which is intended to cure chronic suppuration; but if a simple minor procedure, which can be done in the office or dispensary under local anesthesia, will cure half of the cases so treated, then it has a distinct value and should be further studied.

Another interesting fact presented from the collection of cases is that of the half of the cases reported as not cured about seventy were subsequently operated upon radically, and of these, eighty-eight per cent were reported as cured. Eighty-eight per cent of operations by various persons is a higher percentage of cure than was shown by Dr. Harris; that was probably because the infection was diminished by the previous curettement of the tube. If we have a procedure which can be performed in the dispensary which will cure so large a proportion, and if the cases which are not so cured have a better chance of being cured by the radical operation, then the value of this proceeding is clear, and it should be performed in the office or in the dispensary for all cases that do not require immediate radical intervention.

Only in those cases where this operation fails to cure should the radical operation be done; for if we do not close the eustachian tube, the only thing that is left is to do the radical operation, and you will have to close the tube, anyway; so it seems rational to do this operation first and wait results in all cases where it is allowable to wait before doing the radical.

Dr. Page said that every case has to be decided by itself. There is such a wide difference in these cases, that in some a great deal of deliberation has to be exercised before the radical operation is decided upon. Even when the radical operation has been decided upon and the patient is on the table, it is better, before the middle ear is entered, to investigate the antrum and the mastoid cells and attic region from behind, and to exenterate the tympanic cavity only if the disease lead one to it. For his own part, in patients with a considerable degree of hearing, he had occasionally during the operation decided not to do the complete radical, but some modification of the mastoid operation, and had taken the chance on its stopping the discharge with less effect on the hearing, telling the patient afterward that it might be necessary to operate a second time; and in this way he had not infrequently been lucky enough to get a good result without having to do the more radical operation.

In regard to the hearing, he agreed with what Dr. Dench had said about one being able to give, in the main, a fairly definite prognosis in the three classes he referred to. There is, however, sometimes a gradual loss of hearing for no apparent cause, the patient having at first fair hearing with a dry cavity, and then gradually in a few years losing it. No doubt, all present had had such cases. Even with a clean dry cavity the hearing sometimes gradually gets worse. He had there that evening a girl who was operated upon nine years ago, whose cavity had remained dry all the time, and who still hears a moderate whisper at twenty feet. He knew of one case in which total loss of hearing occurred during the night after the radical operation had been performed.

Dr. Haskin said that during the last twenty-three years he had seen a great many cases that had been operated upon by the radical method in the clinical work in the hospitals, and while he agreed very largely with Dr. Harris' observations, it was not exactly fair to claim that these are true end results, for we do not know what was the history of the cases before, or even after operation. Hospital records are seldom complete, and it is very rare that cases are tested before operation.

In 1908, Dr. McKernon brought out the fact that sometimes for a short while after an operation the hearing appears to be better, but said that his end results, after a few months, was very bad—the hearing almost invariably grew worse. That has been the experience of many. He himself avoided the radical operation until he had employed every possible means of averting it, and had not done a radical mastoid operation in his private practice for ten years. He had not had a case that he had not been able to dry up, and had had no cases with cerebral or cerebellar complications. He believed that if proper precautions were observed, the condition could almost invariably be cleared up; even in clinic cases, he felt that he could cure eighty per cent of the cases without operation. It is a necessary operation in certain cases, but in very few.

Dr. Lederman said he could endorse the statements of the reader of the paper. According to his experience, in recent years the tendency to perform radical mastoid surgery had certainly subsided. This he thought was due to the fact that acute suppurative otitis media is receiving better and more prompt recognition and treatment. Consequently, cases of prolonged suppuration with necrotic changes are less frequent. In former years, in the majority of cases operated upon, the hearing was worse after the radical treatment. He also found that we could not accept the radiograph as the essential factor in determining the actual presence of disease in the mastoid or neighboring structures in these chronic cases. He recalled three instances where the plates showed indications of involvement around the mastoid antrum and middle ear area, and the suppuration cleared up without the radical operation. In another case, where persistent headache and some tenderness existed over the region of the antrum, with the presence of a little discharge in the middle ear, of a chronic nature, the radiograph decided him to perform the radical operation. On opening the mastoid antrum, so little involvement of this cavity and aditus was found that the middle ear was not disturbed, and the local suppuration ceased without further annoyance and with good hearing. Two years ago a boy, eight years old, was referred to Dr. Lederman from out of town for an opinion as to the prognosis of a suppurative otitis of a year's duration. The boy had been under the care of competent and conservative aurists, and after some local treatment a radiograph was taken. The plates suggested a cloudiness in the region of the mastoid antrum; and as a mucoid discharge persisted, the radical operation was advised by the attending aurist.

On examination, Dr. Lederman found a marginal perforation in the anterior superior quadrant, with some mucoid secretion in the canal and middle ear. Bacteriologic examination showed staphylococci. As the patient's tonsils and adenoids had been removed, simple local cleansing by suction and mopping was advised, followed by the application of a specially prepared iodin powder containing ten per cent of iodin, this being promptly liberated as the powder came in contact with the moist mucous membrane. After three such treatments, together with applications of argyrol to the eustachian pharyngeal region, the ear became dry, and has remained so for over a year.

In still another instance where the above treatment was carried out, an equally good result was secured in a young woman patient, who had had a suppuration of both ears for over fifteen years. This patient had been wearing rubber artificial ear drums to improve her hearing, being aware of the existing diseased condition. Both membranes had been absorbed, together with most of the ossicles. Though considerable secretion existed after the artificial drums were discarded, the simple treatment mentioned in the former case, with local application of the iodin powder, brought about a decided improvement, and at present the ears are practically dry, with fair hearing, even with the loss of the normal conducting mechanism.

Further instances of the activity of this new iodin powder will be reported at another time. It has given the same pleasant results under the observation of several colleagues.

Dr. Hays said he did not think it quite right to let the discussion close without mention of the modified radical operation, such as had been presented by Dr. Blackwell a number of times. Many of those who do not perform this operation so often as Dr. Dench, do not get as good results as we would like. There are many cases in which the ears are nonodorous. He had seen a great deal of Dr. Blackwell's work, and his results were excellent; and in many instances he himself had obtained as good results from the modified radical as could have been expected from the complete radical.

Referring to what Dr. Yankauer had said about the eustachian tube, he had some time ago reported a number of cases in which, after six months, nine had remained dry—some of them after a second curetting of the tube. Six were cured originally; the others were sent to Dr. Yankauer for observation, and he curetted them again and they are now well. Many of these cases go away from observation after a short length of time, and we cannot say whether or not they have a permanent good result.

Dr. Blackwell said that something had not been mentioned which must be considered when regarding the statistics reported by Dr. Harris. It is usually true of clinic or hospital cases, where a satisfactory result has been secured, following a radical operation, that the patient is frequently lost sight of and disappears. When he does show up, he insists upon seeing the surgeon who carried him through his former trouble, and regards the advances of all other physicians with a certain amount of suspicion. On the other hand, the cases which are attended by an unsatisfactory result continue to return, and will at times seek to be examined by other physicians, in the hope of obtaining a cure. Only one-third of the total number of cases responded to Dr. Harris' postal of inquiry, and as this small number of cases showed such a high percentage of unfavorable results, it was fair to assume that in the remaining two-thirds not heard from the results were much more favorable.

In regard to the Yankauer operation, as Dr. Dench had said, it is in cases of middle ear discharge of tubal character that one would not recommend a radical operation. Intelligent treatment will dry a large percentage of these ears. Dr. Blackwell said he was convinced that in these cases of tubal discharge, the question at issue was not one of the discharge, but of the hearing. These patients usually hear better when the ear is slightly moist than when it is perfectly dry, and he believed that the ultimate functional result is better when the ear is rendered dry as the result of treatment rather than by obliterating the eustachian tube.

Dr. Richards said that as he understood the matter, the question under discussion was the radical operation itself. As Dr. Harris had seen these cases and examined them with sufficient thoroughness to prepare a paper on the subject, it would be interesting to know if in the unhealed cases the area giving trouble and causing the nonhealing was not, in practically all, anterior to a vertical line dropped through the facial ridge. In other words, was not that portion of the cavity corresponding to the body of the mastoid healed in practically all, and the part giving trouble that which corresponded to the tympanum?

Dr. Harris replied: "Not always, but in a considerable number."

Dr. Richards said that the above was nearly always the case as he had seen it, and that nothing could point more clearly to where the fault was. Why is it that practically all operators can and do get healing in the posterior portion of the cavity, but fail in the anterior portion when they do fail? The reason is plainly this: that the part corresponding to the body of the mastoid is easily accessible, and the dead bone in that area is removed by all operators. Consequently, that portion heals. The front part of the cavity, corresponding to the tympanum, is crowded with important structures; it takes time and patience and work to acquire a technic which enables an operator to remove dead bone from this area without inflicting injury; and those gentlemen who can get but fifty per cent of cases healed are those who do not sufficiently perfect their technic, and who under the circumstances use good judgment in not attacking this area. They would exercise better judgment if they did not operate at all. The defect is in the operator. Why blame the operation?

If we were to go to the representative institutions of this city and watch the radical operations as they are performed one after the other, we would have no difficulty in seeing why it is that the results are as Dr. Harris states. The specific errors most commonly committed are these:

First.—Failure to lower the facial ridge to its absolute limit. Unless this is done, it is utterly impossible to clean out the posterior portion of the tympanum and hypotympanum except at grave risk to the facial nerve. In addition, it prevents proper dressing of the case subsequently, and permits the tympanum to fill up with granulation tissue which effectively blankets the inner tympanic wall, the round and oval windows, thereby decreasing the hearing.

Second.—Failure to remove the serrated fringe of bone representing the anterior margin of the facial ridge back to the descending limb of the facial nerve. This removal should be carried down to the level of the hypotympanic floor, otherwise the hypotympanum in its posterior part is inaccessible and cannot be closed.

Third.—Failure to lower the floor of the bony auditory canal down to the level of the floor of the hypotympanum. Unless these three steps mentioned are taken, it is impossible to get at a mass of cancellated structure posterior to the promontory, which is always diseased in the chronic suppurative cases of the type under consideration, and which if allowed to remain will certainly give trouble. This cancellated structure is peculiarly hard and firm, and shows little tendency to throw itself off by the process of caries.

Fourth.—Failure to shore down the convexity of the anterior bony canal wall so as to increase the anteroposterior axis of the cavity and render (in conjunction with No. 2) the tympanum easily accessible and open. Further, by taking this step we will be enabled—in conjunction with No. 5—to get complete access to the tube area, which otherwise we cannot do. Unless this step is taken in conjunction with No. 2, the tympanum is frequently shut off from the main cavity by a curtain of epithelium which forms during the process of healing, at about the site of the original drum. The tympanum often remains as a suppurating cavity anterior to this epithelial curtain. When this curtain once forms, it is very difficult to eradicate it. If the portion of the cavity anterior to it does by chance clear itself of the suppurative process, the curtain is an advantage, and in such cases we often have excellent hearing. When, however, suppuration continues in the cavity anterior to the curtain, the curtain then is a decided disadvantage. The things which enable it to form are: too small a distance left between the anterior canal wall and the facial ridge, and the leaving of a portion of the annulus tympanicus from which the curtain springs. Little or no packing of the cavity also favors its formation.

Fifth.—Failure to remove the annulus tympanicus completely, and the lip of bone overhanging the mouth of the eustachian tube. When this is done, it is surprising to what a depth we can see into the tube. When this is not done, we cannot curette the tube area, and at the tympanic mouth of the tube there is cancellated bone which is frequently necrotic.

Sixth.—Failure to remove the outer attic wall, particularly in its anterior part, and to carry the removal upward flush with the floor of the skull.

Considering the lack of attention paid to the tympanum in the radical operation as ordinarily performed, the wonder is that as many cases heal as do. The foregoing six reasons account for the vast majority of failures. When these steps are carried out, the cases heal. Dr. Richards said what he particularly wished to emphasize was that if these points are not carried out, the fault is not with the operation, but with the operator.

The chief criticism he had to make on Dr. Harris' paper was that he did not know whether a thorough radical was performed or whether the radical operation was but partially performed in these cases. According to the thoroughness or lack of thoroughness with which we operate, we can present statistics to uphold our contention.

When we consider the high standard of excellence to which the operation has attained, with its splendid results, the fact most clearly brought out by the paper was that there should be such a lack of diffusion of knowledge of the technic required to success among those who have adopted this work as their profession.

Dr. Wesley C. Bowers said that a year ago a stenographer had come to him with an ear which had been discharging for twelve years. The external auditory meatus was narrow externally, but otherwise normal throughout. The ossicles were bound down in the superior quadrant, with the cicatricial membrane apparently adherent to the wall of the middle ear, with a large opening over the tube. The discharge seemed to come from the tube, and he thought it could be helped by local treatment. He treated the ear for three or four weeks, with but little improvement, so he told her that a radical operation would be the best thing to do, though it would probably affect her hearing. She disappeared for a while, but came back two weeks ago and wanted the operation. On pushing the periosteum forward, it was found that there was an opening in the bone of the posterior canal wall which led into a large cholesteatomatous cavity. This illustrates how impossible it is to tell, at times, just what the condition of the mastoid is in the presence of a chronic discharge from the middle ear. Out of the twenty-six radical operations which he has performed in the last year, there were not more than five that did not have cholesteatoma. This being the case, how can we hope to get a permanent cure by local treatment of the middle ear? There would seem to be something wrong about the selection of the cases for this operation.

Dr. T. J. Harris, in closing, thanked the gentlemen who had taken part in the discussion, and said he had brought forward the subject because, as he had stated, he felt that we had been doing the operation long enough to have a frank heart-to-heart discussion of it. He expressed himself as being particularly gratified that Dr. Dench was present, as they had had many discussions in the past. Had Dr. Dench followed his usual practice of reading the paper in advance of the meeting, he would not have made many of the criticisms offered, for he had apparently not grasped them correctly.

So far as the accuracy of the findings was concerned, the number of cases reported was small, but when they were borne out by other cases here and abroad, it formed a sufficient number upon which to base a discussion. In regard to Dr. Dench's statement, Dr. Harris said he had already expressed the opinion that everyone recognized Dr. Dench's great ability in this work, but that this, in his opinion, did not form a criterion for the average results which otologists are getting the country over.

He also wished to state distinctly that his findings did not lead him to condemn the operation; in spite of these findings, the operation is not for a moment to be condemned. What he had attempted to do was to put on record that the findings in this series of cases, in connection with what others had reported to him all over the country, and what he had found in the foreign clinics, made him feel that there is a demand for a much more careful study of cases of chronic suppuration as to whether or not they shall be operated upon radically, than we have been in the habit of giving them. Dr. Page had stated his (Dr. Harris') views accurately.

In speaking of the X-ray findings, Dr. Harris said he meant that this was one of the aids that should be employed; not that we should draw from such findings alone that suppuration meant radical operation; but simply that if we used the X-ray more in determining what cases to operate upon, in connection with other methods, the results would be better.

Dr. Richards had stated many points which he himself had had in mind. No one should jump to the conclusion that in this discussion we were doing more than taking a sober, clear, and candid consideration of the subject. It was true that the work of such men as Dr. Dench, Dr. Richards, and Dr. Page was the level that all should strive for; but the claim made in the past that every case of suppuration was a case for radical operation, and that we should expect a complete cessation of the discharge, was not in accord with his investigations and what he had observed elsewhere.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of November 10, 1916.

Traumatic Rupture of Membrana Tympani Complicating Fracture of the Temporal Bone.

DR. JOHN A. ROBINSON: Two months ago, C. M. C., aged forty-five years, received a blow on the right side of the head. He fell to the floor, striking his head on the left side. He was removed to St. Peter's Hospital in New Brunswick, unconscious, with sharp bleeding from the canal of the left ear. He regained consciousness in three days, remained in bed six days, and was permitted to leave the hospital in a week, and returned to work. He found he could not perform his duties as foreman in a munition plant, because of headache, deafness, discharge from his left ear, and general weakness.

I saw the patient for the first time on September 18th, two weeks after the injury. He then complained of deafness, pulsating tinnitus, and discharge from his left ear, and some occipital headache which was not constant; temperature, 99°; pulse, 64, and some fatigue on exertion. The entire left side of the head was tender, there was slight rigidity of the neck, slight vertigo on turning the head quickly to the right, no nystagmus, no vomiting, no ataxia. Both pupils were equally contracted, but reacted to light and accommodation. The fundus was normal. His mental condition was good.

On examination the canal showed a moderate amount of serous discharge, with slight sagging of the posterosuperior wall. The drum membrane was slightly bulging and reddened, with a linear tear extending through the posterosuperior and posteroinferior quadrant, pulsating and with gaping edges. A smear showed a mixed infection.

Acoumeter not heard; moderate voice, one foot; upper tone limit, 20,000; lower tone limit, 126. Rinné, negative. Weber, referred to the right. No ataxia. Turning elicited nystagmus for ten seconds.

The patient was admitted to the New York Eye and Ear

Infirmary, and an X-ray plate, made by Dr. George S. Dixon, showed both mastoids to be of pneumatic type, the right clear, the left hazy to cloudy. There were two lines of fracture, one about three centimeters long, curved somewhat, and directed upward and backward on the left parietal, directly above the mastoid process. The other was about four centimeters long, not connected with the first, but beginning a little behind it, and running downward and forward through the posterior portion of the squama, and ending in or near the mastoid antrum.

The patient was put to bed and wick drainage begun, the gauze being changed four times in the twenty-four hours, after cleansing the canal. It soon became apparent that the discharge was intermittent; at times the gauze would remain dry for some hours, and again would become saturated in half an hour. A small quantity of the discharge was examined, and as it did not respond to the test for glucose, cerebrospinal fluid was ruled out. It reacted, however, as blood serum. The inference was that the left mastoid was more likely filled with blood than pus.

The patient was admitted on September 18th, and remained in the hospital ten days, spending seven days in bed. The discharge gradually ceased, and the ear became dry in ten days. At the dressings the first four days after admission the serous discharge was profuse. The headache gradually ceased. At times there would be slight vertigo on turning quickly to the right. On September 30th, four weeks after the injury, the rupture in the drum head closed. The hearing has steadily improved. He now hears a low whisper at six feet.

DISCUSSION.

Dr. Phillips said that all who had had much experience could relate interesting histories of this kind. In one instance that he recalled a man fell from a ladder—he was a carpenter—and struck on his head. He was unconscious for several days, and had a hemorrhage from both ears, having sustained a traumatic rupture of both membranes. After about two weeks consciousness returned, but he was totally deaf in both ears. Dr. Phillips said that this was the only bilateral case he had seen.

Dr. Danziger inquired whether there was any danger of traumatic epilepsy after such a fracture. He had seen, about a year ago, a case of fracture of the occipital bone extending forward through the external meatus. The patient had a hemorrhage from the ear. The labyrinth was tested and found normal; the hearing was normal; the tuning fork was lateralized to the affected ear; there was no vertigo or nystagmus. The blood clot in the ear was not disturbed.

Within a few days the patient felt normal, but an X-ray picture by Dr. Caldwell disclosed the above described fracture. The surgeon who saw him afterward thought of the possibility of traumatic epilepsy later in life, in spite of the fact that the motor area was not involved by the fracture.

Dr. Voislawsky, replying to Dr. Danziger, said that he had had a case in which epilepsy did follow, but it lasted for only eighteen months; and as he had not seen or heard from the patient for three years, he had every reason to believe that the condition had cleared up.

Paper: A Case of Labyrinthine Fistula With Complete Loss of Cochlear Function and Persistence of Normal Vestibular Function.*

By Julius Auerbach, M. D.

DISCUSSION.

Dr. Braun said that Dr. Auerbach's case was a most interesting one, but was not so very unusual. That the hearing was completely abolished and the static labyrinth still functionating, was due to the fact that the end organs in the cochlea are less resistant to infection than those in the vestibule and semicircular canals. Alexander had the opportunity of examining, histologically, several cases of acute suppurative labyrinthitis, which died early in the disease. He found the organs of Corti destroyed, and the cristæ and maculæ still intact. Dr. Auerbach's case, in all probability, had had a serous labyrinthitis, seven months ago, which had destroyed the end organs in the cochlea, but had left the vestibular end organs intact.

Dr. Braun had not had an opportunity of examining the patient's middle ear, but it did not seem likely to him that an inflammatory process which was so severe as to cause a destruction of a portion of the bony labyrinthine capsule, had entirely healed spontaneously. He thought it would be much safer, in a case of this sort, to do a radical mastoid operation to insure healing. He had operated on nine or ten cases of labyrinthine fistula, and all had healed. He did a radical mastoid, and did not touch the labyrinthine fistula. The fistula cicatrized over, and all symptoms disappeared.

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*See page 117.

Dr. Phillips was inclined to agree with Dr. Braun about the present condition of the case. He had looked into the ear, and the intertympanic cavity well up in the attic region was covered with inspissated pus, and he was sure a large crust could be removed at this moment. The thing that had saved the man from great discomfort was no doubt the breaking down of the outer wall of the attic and the sloughing of the bone, so that fair drainage was maintained. It would almost seem as if the discharge came from around the region of the fistula. If this was an instance of fistula in a chronic case that was better for not being submitted to the radical mastoid operation, it was the first of the kind that Dr. Phillips had seen. He was much inclined to think that even yet there may be some trouble, for he believed that there was still some cholesteatomatous material in there.

Dr. Braun, in reply to a request from Dr. Harris, as to what the pathologic condition was in the fistula cases, said that in all the cases he had had, where there was a fistula reaction, he had found a fistula in the horizontal semicircular canal. It varied in size, sometimes being no larger than a pin point, and at other times a quarter of an inch in length, and the entire width of the canal. In several cases, in which a fistula was found at operation, and in which there was no fistula reaction, the location of the fistula was in the cochlea, or on the superior or posterior semicircular canal. The fistulæ all occurred in chronic suppurative middle ear conditions. The mastoid cells were sclerosed, and the antrum and middle ear contained pus and granulations, and in some instances cholesteatoma.

Dr. Phillips said he thought the vast majority of these cases had cholesteatoma.

Dr. Danziger asked if Dr. Braun had ever seen a partial recovery of either the cochlear or the vestibular function after an unquestionable attack of serous labyrinthitis. He himself had never seen a recovery from an attack of serous labyrinthitis without complete loss of function. The patient's loss of function of the cochlear apparatus is due to a process of atrophy. When this patient had his attack of vertigo, he probably did not have a labyrinthitis, but some disturbance of the air pressure or circulation acted on the vestibular apparatus more vehemently on account of the fistula, which may be due to some former bone process or even a natural dehiscence. He thought the patient should not be subjected to any operation.

Dr. Braun, in reply to Dr. Danziger, said he had seen several cases of serous labyrinthitis in which the vestibular function was retained during the course of the disease.

An Extensive Cholesteatomatous Mass Simulating a Sequestrum in the X-Ray Plate.

Dr. J. J. King: Case 1.—H. C., a normally developed youth of eighteen years, was admitted to Dr. Lewis' service in the New York Ear and Ear Infirmary, on July 15, 1916, with the following history:

He had had the ordinary diseases of childhood. Eight years ago, after scarlet fever, he had an abscess in the left ear, and it has been discharging at intervals ever since. At times there would be swelling and pain over the mastoid tip. The hearing was defective.

Examination revealed an occlusion of the exterior bony canal to such an extent that nothing larger than a fine applicator could be passed. There was swelling, redness, and a fistulous tract just over the tip of the mastoid. A very foul and purulent, but scanty, discharge came from the canal and this fistulous tract. Upon probing through the fistula, bone could be felt. The patient could hear a watch and acoumeter in this ear only upon contact.

The X-ray plate showed a large shadow in the mastoid, and Dr. Dixon thought this shadow indicated a sequestrum—in fact, this was the chief point of interest in the case.

On July 28th the radical operation was performed in the usual way, under general anesthesia. Upon retracting the flaps a cortical perforation was found, and almost all of the mastoid cavity was found to be filled with cholesteatoma. This mass had destroyed nearly all the cells, and when it was removed the wound had the appearance seen after a simple mastoid had been performed. The wound was closed with metal clips, and the patient made an uneventful recovery.

Otitis Media Purulenta Acuta, Mastoid Abscess With Perisinus Abscess, Which Was Shown in Radiograph.

DR. J. J. KING: Case 2.—A. F., female, was admitted to Dr. Robert Lewis' service at the New York Eye and Ear Infirmary on September 26, 1916, with the following history:

One month previous to admission to the hospital she had a severe nose bleed. She then blew her nose and a sudden noise was noticed in the ear, followed by a severe pain. She had had no trouble with the ear prior to that time. A week later the left ear began to discharge, and the discharge has been continuous and profuse ever since. Two weeks ago the pain and swelling became severe over the mastoid; no vertigo; occasional nausea.

Examination upon admission to the hospital revealed a profuse discharge from the middle ear, with a red and edematous drum membrane. There was some swelling and edema over the mastoid, with exquisite tenderness over the region of the mastoid emissary vein.

The temperature on admission was 102.6° F.; pulse, 134.

X-ray findings by Dr. Dixon: "The right is small, typical, pneumatic. The left is also pneumatic, with elongated tip. A large and very peculiar mastoid emissary vein; it is very cloudy."

After studying the plate, Dr. Dixon advised immediate operation. A large shadow just below the mastoid emissary vein was shown in the plate, and this proved at operation to be a perisinus abscess.

On September 28, 1916, the usual postauricular incision was made and a simple mastoidectomy performed. The cortex was not perforated and was normal in appearance. Much free pus was found in the antrum, and granulations were present in the cells. The entire mastoid tip was necrotic and was removed en masse. A large perisinus abscess was found just below the mastoid emissary vein, and in removing this abscess a large area of sinus was exposed. Below the tip of the mastoid a very large abscess was found in the muscles of the neck, from which a large quantity of pus came. The dura was exposed in three small areas, from which hemorrhage of a dark color was free. The wound was packed with iodoform gauze and partly closed with metal clips.

After this operation the patient was more comfortable and the wound progressed normally, but there was still some pain on pressure posterior to the wound. The temperature ranged from 99° to 102° F., going up every afternoon. It was a regular septic temperature.

On October 10, 1916, after consultation with Dr. Blackwell, the wound was again opened, and an incision was made extending backward and downward for one and one-half inches behind the edge of the wound. A large abscess was found and much pus was evacuated. A counter puncture was made below, in the most dependent part of the abscess, and through and through drainage was established by means of a cigarette drain.

After this operation the temperature rapidly subsided to normal. The healing of the wound was satisfactory, and the patient's recovery was uneventful in every way. She was discharged from the hospital, cured, on November 4, 1916.

DISCUSSION.

DR. GUTTMAN inquired whether the case was a typical Bezold mastoid, to which Dr. King replied in the negative.

DR. HURD told of two cases of extensive cholesteatoma in which the labyrinth was invaded.

DR. HELLER said that Dr. King's first case reminded him of a young girl of fourteen or so who had had scarlet fever when two years old, and was operated upon for acute mastoiditis. Thereafter she had had no medical attention whatsoever; nothing but the usual household remedies. When the discharge got bad enough to offend her or the family, she syringed it. The ossicles were gone, and the patient had a foul discharge; but she applied for the treatment of a lump behind the ear, the size of a hen's egg, with a bulging of the scar. As the condition was absolutely painless, it was evidently not an inflammatory condition. No cholesteatoma was visible in the tympanum or meatus, and the condition suggested a sebaceous cyst in an unusual location. She consented to an operation, and at first he thought he had found a sebaceous cyst; but on opening the capsule he found a large mass of sebaceous-like material, which proved to be a cholesteatoma. The cortex underneath this was hard, and a hole not much larger than the lead in a pencil was disclosed, communicating with old mastoid cavity. This was enlarged, and another cholesteatoma was found as large as the first. It made a sort of hourglass shaped mass. The inside was completely reamed out, as Dr. Hurd had described. All that remained to be done was to take down the posterior wall, and the mastoid operation was complete. Recovery was uneventful.

DR. BLACKWELL, referring to Dr. King's case of cholesteatoma, said that it had been his experience that these cases uesually recover after operation, despite their extensive involvement. This was probably due to the formation of a limiting capsule of rarefied bone about the cholesteatomatous and abscess cavity, no doubt caused by the extreme chronicity of the infection, this delimiting wall protecting more or less the important intracranial tissues.

In the case of perisinus abscess, he wished to refer to the apparent diagnosis from the X-ray plates made by Dr. Dixon. Dr. Dixon's skill and technic in making and reading these plates has certainly reached an unusual degree of accuracy.

DR. SCRUTON said that he was not familiar with the history of Dr. King's second case, but that such a case is sometimes the forerunnner of an atypical sinus thrombosis. Dr. King said that the temperature was approximately normal, but on the premise that it is still running a temperature, if he were managing it he would feel that it might develop into a septic sinus thrombosis. One can have a case of that kind with a temperature of 100° or 100.5° for weeks. The patient would not be sick, but would have a little headache or a little tenderness of glands in the neck. The only thing that would absolutely point to the condition, in addition to having had a possible injury of the sinus, would be a leucocytosis of 18,000 and a high polymorphonuclear count. He suggested that if a leucocytosis examination had not been made, it should be done.

DR. DURKEE told of a case that showed how these cases of abscess in the tissues of the neck may at times be rather puzzling. The patient, a man of fifty years, gave a history of pain behind the ear which had continued for about two months, when there was a discharge from the ear and a large swelling over the upper part of the mastoid. This swelling disappeared in a few days, and at the same time a swelling appeared in the upper part of the neck. When the man first came into his hands there was a large fluctuating swelling in the upper part of the neck and over the lower part of the tip of the mastoid. There was no swelling over the upper part of the mastoid, and no tenderness over the antrum or emissary vein. The canal showed a large amount of pus, and there was sagging of its upper wall. The abscess in the upper part of the neck was opened and a sinus was found extending from its upper part and underneath the periosteum to a necrotic spot about the size of a ten-cent piece in the bone over the antrum. The usual mastoid operation was done. The bone was gone over the sinus and the dura above, and both sinus and dura were covered with granulations. Some very deep cells were found below the antrum between the sinus and the posterior canal wall. These were uncovered, but not curetted, for fear of injuring the facial nerve. At the first dressing the entire wound was in good condition except the deep cells that were found below the antrum. From these there came a large amount of pus. In irrigating these cells the solution came into the patient's throat. In two or three days deep fluctuation was found low down in the neck below the sternomastoid, and a deep abscess was opened. The opening in the pharynx closed in a few days, and the mastoid wound was healing well, but the patient developed erysipelas and died.

This case shows how a case of superficial abscess in the neck, without a good history of the ear condition and a thorough examination of the ear, might lead to an error in diagnosis.

Dr. Voislawsky asked if Dr. Durkee had found how the fluid got into the throat, to which Dr. Durkee replied in the negative.

Dr. Guttman said that all have cases where, after a mastoid operation, the temperature keeps up, and after looking for the cause and not thinking of sinus thrombosis, redness is found below the tip of the mastoid, and an abscess developing which sometimes breaks through into the sinus and causes a pharyngeal abscess. This seemed to be the case in this instance. It was not probable that in the case described the pus should have attacked the sinus by breaking through the hard ivory-like bone covering the sinus, when it was easier for the pus to burrow into the sheath of the muscle. Therefore, he did not think it was a case of sinus thrombosis.

Dr. Voislawsky said that Dr. Scruton's suggestion was worthy of consideration.

Dr. King expressed his appreciation of the full discussion of his cases. The temperature in the second case never suggested a sinus thrombosis temperature. With the exception of once, it never went above 102°, and then it was 103°. The patient ran that temperature right along. She never had any steep temperature; after the second operation, it went right down and remained down. The wound has completely healed now, and the patient reports occasionally for observation. Dr. Scruton's suggestion of a sinus thrombosis, however, was well taken, and we should always be on the lookout for thrombosis in atypical cases.

Relation of Air Pressure in the Ear to Ear Diseases.

Dr. John W. Durkee endeavored to prove whether true or not the statement often found in the literature of otology, that back of a nasal obstruction, in the posterior part of the nose and nasopharynx, and in the eustachian tube and middle ear, there is, during inspiration and during the act of swallowing, a negative pressure that is greater than that in the normal nose, and that this increased negative pressure is a common cause of ear disease, especially chronic catarrhal otitis media.

An effort was made to determine the air pressure in the normal nose. Ten patients with normal noses were chosen and, using a water manometer connected by rubber tubing with a straight eustachian catheter, records were made from the middle of the inferior meatus and nasopharynx.

To determine the air pressure back of a nasal obstruction, ten patients, in whom there was a deviation of the nasal septum which practically closed one side of the nose, were chosen, and records made behind the deviation and at a corresponding place on the clear side of the nose.

Seven of the patients with normal noses were then chosen, and one side of the nose closed with cotton while the other side remained clear. Records were made behind this obstruction and at a corresponding place on the unobstructed side.

An average of these measurements gave the following figures:

Normal nose, at the middle of the inferior meatus, plus 2.5 millimeters of water and minus 3.4.

Cases of deviation of the nasal septum: behind the deviation, plus 3.4 and minus 4.5; on the unobstructed side, plus 4.2 and minus 5.2.

The cases in which one side was closed with cotton: obstructed side, plus 3.5 and minus 5.0; on the clear side, plus 4.3 and minus 6.4.

A combination of the deviated septum cases and the cases in which one side was closed with cotton: obstructed side, plus 3.4 and minus 4.7; on the clear side, plus 4.2 and minus 5.8. During swallowing the pressure in the nasopharynx was zero.

The conclusion reached was that the negative air pressure found in the nose and nasopharynx in cases of nasal obstruction can hardly be considered a cause of ear disease.

DISCUSSION.

DR. HARRIS said that to him a paper like this, which had nothing to do with surgery, was delightfully refreshing. It makes one remember that there are many problems in otology beside surgical ones. Dr. Durkee was to be congratulated on the very careful work he had put into this paper; it was the sort that all like to hear. There are two kinds of original work. Very few men are capable of evolving something entirely new. He had heard some men say that when they thought they had discovered a new idea they found it had been patented long ago. There is a large field for such work as Dr. Durkee had done—in the proving or disproving of certain assertions that had been previously made.

Dr. Durkee's method of procedure was decidedly ingenious and interesting; and those who had not had opportunity to do this kind of work would find it difficult to take exception to his conclusions. It would require personal work of a similar kind to discuss the paper, but the conclusions which Dr. Durkee had reached were in line with what Dr. Harris himself had always felt.

The recurring question of the effect, or the possible effect, of negative pressure on the middle ear has not been settled, and may not ever be; but certainly the old proposition that we have in the ear a membrane with atmospheric pressure on the outside and inside, and if we have a tube which is obstructed, the drum will be driven in, while it obtains sometimes, does not always obtain. All clinical experiences show exceptions to this. We see drum membranes that are not retracted although the tube is obstructed; and we see every day tubes that are apparently normal, and yet the drums are retracted. Dr. Durkee's conclusion that the tube is of such a character that we cannot arrive at a fixed and firm conclusion seemed just and fair. In the question of negative pressure there are many factors entering into the problem, and the relation of nasal disease to diseases of the middle ear is still far from settled. The very broad and strong statement which we have heard, and still hear, that all that is necessary to cure middle ear disease is to remove obstructions from the nose, has been shown to be wrong. There are many other factors entering into middle ear disease.

Dr. Harris said that he personally was inclined to believe that often bacteria are the cause, and that in many instances we have to look outside of the nose for the cause of disease existing in the middle ear. It would be interesting if Dr. Durkee's experiments, which were necessarily limited in number, could be repeated.

Dr. Hubby also congratulated Dr. Durkee on his painstaking work. It was certainly very important to take up such questions as these. He agreed with Dr. Durkee that in a one-sided nasal obstruction the negative pressure in the nasopharynx, during swallowing and inspiration, is not sufficient to produce any change in the middle ear directly. On the other hand, if both sides are obstructed, Dr. Durkee found that the negative pressure on swallowing increased less than a millimeter. Although this loss of a millimeter sounds very small, yet it is sufficient to produce a sense of fullness in the ear when you try it, if you have a normal eustachian tube, and it is also sufficient to be demonstrated, as has been done with the manometer by Politzer and others. Of course, as Dr. Durkee said, chronic bilateral obstruction is not a condition that is common or allowed to continue. One must remember also the fact that during swallowing the eustachian tube opens, so that the pressure of less than a millimeter is readily communicated to the middle ear.

Taking up the secondary effect of changes in pressure in the nasopharynx, positive and negative, it is quite a common experience in chronic turgescent rhinitis to find that the turgescence shifts from day to day, attacking first one side and then the other; and it is believed that this is due to changes of air pressure on one side when the other side is obstructed—a to-and-fro compression and suction exerted on the nasal mucosa; and if this is maintained for any great period of time, chronic hyperplastic rhinitis results with involvement of the eustachian tube and tympanum as sequelæ.

Dr. Glogau said that he was very much interested in Dr. Durkee's experiments, for he himself had been doing some similar work at the Speech Research Laboratory of Vanderbilt Clinic for the past two years. Dr. Durkee's method is very interesting. It is similar to the one that had been used by Gutzmann in Berlin and by Froeschels in Vienna. Smoked paper records for measuring the air pressure within the nose and nasopharynx have been in vogue for a long time, although usually Marey's tambour has been used. Dr. Glogau found that when a small hole has been cut into the rubber tube near the nasal olive the nasal air pressure is reduced to such a degree as to render the oscillations of the lever comparatively small. In this way any obstruction to normal breathing can be easily recorded. Dr. Glogau, in different papers, had advocated that the rhinologist should make smoked paper records of nasal breathing before and after nasal operations, whereby both the doctor and the patient could be objectively convinced of a return to the normal of formerly abnormal breathing.

Dr. Durkee said that he made his experiments on people that were breathing normally. In order to find out whether or not a person breathes normally, breathing curves of the thorax and of the abdomen must be recorded first. For we have certain invariable rules for normal breathing, both during rest and during speech. During rest, the length of the wave for inspiration and expiration in both thorax and abdomen is almost the same, the expiration being only slightly longer. The breathing curve of the nose is somewhat irregular, but shows also the same length of the rising and of the falling part of the curve. The moment we start to speak, the nasal curve becomes a straight line, elevations being only noticeable at the pronunciation of the letters m, n, and ng. The thoracic and abdominal breathing curves during speech differ also considerably from those during rest. The inspiration becomes very short, the expiration markedly prolonged. The quick inhalation goes through the mouth, and we speak on a prolonged exhalation.

In persons suffering from speech defects, the breathing curves are also abnormal. In pronounced stuttering, for instance, even the curves of mere breathing show irregular elevations and sudden jerks. During speech the thoracic and abdominal breathing curve of the stutterer become pathognomonic. We find an irregular conglomeration of inspiratory and expiratory jerks, interrupted by straight lines, indicating the stoppage of breath. The nasal curve of the stutterer during speech shows characteristic fluctuations, due to the extension of the irregular concussions of breath to the soft palate, and from there to the air space within the nose.

It is quite frequently overlooked that a normally functionating soft palate is essential for the aeration of the middle ear. Within the soft palate are contained the opening and closing muscles of the eustachian tube. Insufficiencies of the soft palate can be easily recorded on smoked paper. By including such experiments as Dr. Durkee reported, and also the broader field of experimental phonetics, the specialty of otolaryngology will broaden its scope.

Dr. Hays said he had hoped that Dr. Durkee would give some new facts which would be helpful in explaining why a certain atmospheric pressure is necessary in order to have a normal ventilation of the eustachian tube. When we examine a nasopharynx in these cases, we often find a number of pathologic conditions. In most of the cases of catarrhal deafness the trouble lies in a diseased condition of the eustachian tube which is usually secondary to some pathologic condition in the nose and throat. Whether the pathologic condition in the nose is directly accountable for the impaired hearing, depends upon the individual case. But the fact remains that in almost every case of catarrhal deafness we find a stenosed or an atrophic eustachian tube. It has been stated tonight that in every normal act of swallowing we manage to get a certain amount of air into the middle ear, which is sufficient to counteract the pressure outside the drum, so that we have an equal pressure on both sides. Whenever this atmospheric pressure is interfered with, we get a change in the normal ossicular transmission of sound. The best evidence that air pressure is necessary is shown by the change in hearing when the Valsalva method of inflation is used. Here an exaggerated air force enters the middle ear, which distends the drum, and increases the hearing for the time being. Incidentally, Dr. Hays said, such a method of inflation is inadvisable, for sooner or later the drum becomes relaxed and sound waves do not reach the cochlea.

Regardless of what others might say, Dr. Hays said that he had seen many cases of deafness associated with pathologic conditions in the nose and throat. We are wont to be careless in our examination of the nose and throat in ear clinics, but in private practice, where examinations are more thorough, it is surprising how often we find abnormalities which not only need correction, but which prove by the increased hearing that such correction has been the basic fact in the trouble. His explanation is that proper tubal ventilation is established. Some years ago he investigated a number of cases of deafness, paying particular attention to nose and throat abnormalities, and in a number of these cases nose and throat operations were performed and an improvement in the hearing was noted.

Dr. Hays concluded that physiologic experiment and pathologic conditions could not be compared, and that each case had to be looked at from its individual standpoint.

Dr. Scruton said that Dr. Durkee had showed that air pressure in the pharynx has very little to do with the ear conditions. That is borne out clinically by the fact that most of the nasal obstruction cases, deviated septum, etc., do not complain of ear conditions; this statement refers specifically to adult cases. Examination day after day in the ear clinic of cases of otitis media purulenta chronica indicates that perhaps one-half have apparent nasal obstruction, and a majority of these are compensated.

Dr. Durkee, being asked to close the discussion, said that he had nothing to add.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of December 8, 1916.

Case of Serous Labyrinthitis Complicating Acute Otitis Media.

Dr. Ernst M. Danziger: The patient, Mrs. M., was taken sick with grippe during the month of April, 1916, and in the beginning of May developed an acute attack of otitis media. She suffered for a week before calling in Dr. Danziger. She then had a temperature of 101° and a typical picture of acute otitis. On the second of May, in the morning, a paracentesis was done, followed by an immense amount of serum oozing through the cut. The patient felt almost immediate relief until in the evening, ten hours later, when she felt sick at her stomach. When seen at that time she had a typical nystagmus toward the affected side, but could hear with the noise apparatus in the other ear. The caloric test was not made, for the patient was too sick. A diagnosis of serous labyrinthitis was made, and the patient was put to bed in a dark room, and so placed that when she looked up the nystagmus would not occur. The next day the symptoms were the same, but the nystagmus had changed from the affected to the healthy side. She still heard with the noise apparatus on the other side. In another day she got totally deaf, and continued in that condition for a week, and then got better. On November 1st she was put in a turning chair and turned, first to one side and then to the other, showing a shortened after-nystagmus. The affected ear did not respond to any caloric test, and there was no nystagmus present; the patient could walk without dizziness, and showed no disturbance of the vestibular apparatus. She was totally deaf, however, with the Bárány apparatus to the good ear. The reading test showed the typical rising voice with the noise apparatus. Lateralization of tuning fork to the good ear.

Dr. Danziger said that this was the fifth case of the kind that he had seen in his experience, and the picture and the result were so typical, he thought that the complete loss of function was a part of the picture of diffuse serous labyrinthitis. He had not seen a single case recover with hearing on the affected side, and he did not think one should expect recovery. He had looked up the literature, and had asked various men if they had seen such recovery, and they had all replied in the negative.

DISCUSSION.

Dr. Braun said that he had a similar case last June. A man of about thirty years had a double acute otitis for two weeks. He had had a paracentesis twice in both ears. When seen by Dr. Braun the left ear had healed. The right drum membrane had closed up, was red and bulging, and there was a slight amount of tenderness over the mastoid. Another paracentesis was done on the right drum membrane at about five o'clock in the afternoon. At two o'clock the following morning he had dizziness and vomited. When seen at ten o'clock in the morning he had been suffering from dizziness for eight hours, and had a typical picture of an acute labyrinthitis, with complete destruction of function. He had nystagmus toward the sound side, and was completely deaf in the affected ear. No caloric test was done because he was too sick. His temperature was normal. Dr. Braun said he did not then know whether it was serous or a suppurative labyrinthitis, and does not yet know. The severe symptoms went on for a week and then subsided; and in ten days the nystagmus had disappeared. Today the patient is well, excepting for a dead labyrinth on that side and a slight amount of vertigo on sudden movement. One could not say in either this case or Dr. Danziger's case what the pathologic lesion was, but only that it was an acute labyrinthitis. These cases do not come to autopsy, so they cannot be examined pathologically. Complete loss of function may occur in both forms of labyrinthitis.

Dr. Danziger corroborated what Dr. Braun had said. If a case comes to autopsy, one knows what was going on, but these cases which are regarded as serous labyrinthitis do not recover with any function.

In reply to a question from Dr. Eagleton, Dr. Danziger said: "At a very early stage of an otitis the probable diagnosis of a serous labyrinthitis can be made. An acute labyrinthitis following later, when changes in the bone have taken place, gives the strong suspicion that we have to deal with a purulent labyrinthitis."

Tubercular Mastoiditis With Sequelæ—Multiple Operations.

DR. WILLIAM LEDLIE CULBERT said that he had been asked to present this patient, a full report of whose case had been made at the annual meeting of the American Laryngological, Rhinological and Otological Society at White Sulphur Springs last May. This report was published in *The Laryngoscope* of December, 1916. The members could see for themselves what a healthy looking, unusually robust child the patient now is. He then gave the history of the case briefly, as follows:

On March 22, 1914, the child, then seven and a half months old, was brought to him with a history of having had a discharge from the right ear for six weeks, accompanied with more or less severe pain. During this time she had developed a beginning facial paralysis. She was very much emaciated, had a temperature of 101°; pulse, 144; respiration, 24. Chest and abdomen negative. The left ear was normal; right, otitis media purulenta acuta, with complete sagging of the external canal wall. There was a purulent discharge, but inadequate drainage. The tissues around the external ear were infiltrated, producing an extensive diffuse furuncle. There was a purulent discharge from the nose.

The same day a simple mastoid operation was done. The whole mastoid was found to be involved; it was thoroughly cleaned out and the furuncle was incised. Later, the stitches of the mastoid wound all pulled out and the wound did not granulate, but assumed an unhealthy grayish appearance. The von Pirquet test gave a strongly positive reaction, and washings from the mastoid wound demonstrated the presence of tubercle bacilli.

On April 8th tuberculin injections of one minim of a 1 to 1,000,000 dilution were begun, and these were gradually increased in strength at five-day intervals for a year, until ten minims of a 1/10,000 dilution were given. During a part of this time there was a large amount of pus coming from the parotid gland, and from the other wounds the whole time.

On May 20th the child was again sent to the hospital for the incision and drainage of the furuncle. After that the right cervical glands became involved and developed into a large fluctuating mass. On July 16th she was sent to the hospital for the third time, and this mass of purulent glands was dissected out.

In March, 1915, the staphylococcus hemolyticus was demonstrated from the middle ear, and an autogenous vaccine was prepared and administered through April and May. This had a marked effect on the copious purulent secretion from the various wounds, reducing the discharge to practically nothing, and also reducing the purulent nasal secretion. During these months the child gained steadily in strength and health—indeed, became quite robust.

In July, 1915, the child came back again with a temperature of 105.2° ; pulse, 137 ; respiration, 44. There was also a discharge from the middle ear, which had come on suddenly. She was immediately taken into the hospital and a radical mastoid operation was performed. A sequestrum was found, involving the whole of the mastoid process with the posterior canal wall. This sequestrum was removed en masse, and the subsequent pathologic report showed it to be typical tuberculosis of the bone. From that time (a year and a half ago) the child made an uneventful recovery, and has remained well ever since. During the healing process another washing was made from the wound and examined for tubercle bacilli, but none was found. The ear has been perfectly dry for a year and a half.

DISCUSSION.

DR. QUINLAN asked whether the incision was deep and extensive, involving the facial nerve which was buried in the substance of the parotid gland.

DR. CULBERT replied in the negative.

DR. QUINLAN asked if a Wassermann test was made, and upon Dr. Culbert's replying in the affirmative, he said that the presence of the tubercle bacilli would almost eliminate that, but he had frequently seen in these conditions a mixed infection, viz., tuberculosis and syphilis.

DR. DWYER said he thought this was the first case on record in which the tubercle bacillus was isolated from the ear and obtained in a pure culture on media, according to the method described in a paper last year. In this case they had the demonstration of the organism in the smear, the growth in pure culture, the von Pirquet reaction, and the finding of tubercular disease in the sequestrum. According to his statistics, many of the cases of mastoiditis and otitis media are tubercular in such children.

Dr. Dwyer, replying to Dr. Quinlan's query about the Wassermann test, said that that test was made as a routine procedure in all these cases, and that it was negative.

Dr. Danziger asked if the occurrence of the facial paralysis during the acute otitis would not have been one indication for a more radical operation in the beginning.

Dr. Braun, replying to Dr. Danziger's question, said that a facial paralysis occurring during an acute middle ear suppuration is not an indication for operation. He had seen a dozen such cases which all recovered without operation. The cause of facial paralysis during an acute otitis is in most instances an anatomic abnormality, a dehiscence in the bony wall of the facial canal in its intratympanic portion. When facial paralysis occurs in chronic middle ear conditions, it is due usually to bone necrosis, and is an indication for operation.

The point that had impressed him in Dr. Culbert's case was that tuberculosis in the ear seems to act differently in children from what it does in adults. In children it is very virulent, and is usually attended with extensive complications, and many cases have been fatal, whereas in adults, tuberculosis of the middle ear usually runs a fairly benign course. It is very obstinate to treatment in adults, but rarely causes any serious complications.

Dr. Guttman asked whether the findings of the washing were pathognomonic for tubercle bacilli; for all of these complications ceased as soon as the sequestrum was removed.

Dr. Dwyer said he thought this case was the first one on record in which the diagnosis had been made by the isolation of the tubercle bacillus from the ear in culture. The diagnosis was complete from start to finish—tubercle bacilli in the smear from the ear washings; tubercle bacilli isolated in pure culture; von Pirquet reaction positive (quite reliable in a child of this age); and the final proof, microscopically in the tissue.

Replying to Dr. Guttman's inquiry, he said that this was probably a localized tuberculosis of the ear, as the examination was otherwise negative, and localized tuberculosis is common enough for us to justify such a diagnosis.

DR. CULBERT, closing the discussion, said he had nothing to add, excepting to respond to the question in regard to doing a radical operation during an acute condition. No one would think of performing such an operation on a child only seven months old, no matter how aggravated the condition. One would feel that he would at least give the child a chance with the simple operation. Again, following the first operation, the facial paralysis did subside—he had neglected to state that—some five weeks after the first operation, with the bad furuncular condition which was being treated. The parotid then became involved, and there was a large parotid abscess. It was then that the facial paralysis was again noted, and in cleaning out a small opening was made, and the necrotic tissue very carefully curetted out. The wound was packed with gauze wet with a one per cent formalin solution, and it healed kindly after that procedure. The facial paralysis was at its worst when the child had the parotid abscess. Since then it had gradually become less and less, and the child can now partly close the eye. The mother says that when the child sleeps and is relaxed the eye comes together. How much of the power of the nerve will be regained is a matter for the future to decide.

Paper: Case of Salivary Fistula Following Simple Mastoidectomy With Cervical Abscess.*

By F. C. SCHREIBER, M. D.

(By Invitation.)

DISCUSSION.

DR. KING congratulated Dr. Schreiber upon the excellent work he had done in working up this case, and thanked him for reporting it before the section. Dr. Schreiber has covered the ground so thoroughly in his presentation that little else can be added in discussion.

DR. SCHREIBER, in closing, said that fistulæ affecting the parotid gland are very infrequent, particularly those involving the parenchyma incurred by faulty technic during a mastoidectomy. Incision of abscesses of the parotid are seldom followed by a fistulous tract. There is little doubt that the gland is often injured by sharp retractors, but nature seems provident and closes over the injury.

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*See page 113.

Dr. Schreiber, in reply to an inquiry from Dr. Eagleton as to whether he was going to try to close this fistula, answered in the affirmative. Dr. V. Hammond of Boston had reported a similar case in 1915, but he had made no chemical analysis of the secretion.

Paper: Results in Four Cases of a Modified Radical Operation for Chronic Purulent Otitis Media.*

By HUGH B. BLACKWELL, M. D.

DISCUSSION.

DR. BRAUN asked to what Dr. Blackwell ascribed the improvement in hearing in the first case. He had understood Dr. Blackwell to say that this patient was completely deaf before the operation. Complete deafness is never due to middle ear disease. No matter how much destruction there is in the middle ear, there must be some hearing unless there is involvement of the cochlea or cochlear nerve.

DR. BLACKWELL, replying to Dr. Braun, said that the marked deafness existing previous to operation in the first case, associated with a normal vestibular reaction, and the return of hearing to the affected ear after the operation, was to him the most interesting feature in all the four cases presented. He said that he had never seen such a marked loss of hearing in an otitis media purulenta chronica with subsequent restoration of function, and that he could offer no pathologic theory to account for the clinical facts in the case.

DR. WELLS P. EAGLETON reported a case of

Metastatic Carcinoma of the Temporal Bone.

DISCUSSION.

DR. LEWALD said that he had made two X-ray examinations of two cases of metastatic carcinoma of the skull, one showing multiple carcinomatous deposits in the skull, following the removal of the breast for carcinoma, three years previously. Professor Wood also saw this case, and thought there must have been an involvement of an artery supplying the skull, with the result that groups of cancer cells were thrown into the branches of the artery, causing the deposit of a great number of cells in the bone. The other case was one of a single metastatic carcinoma of the vault of the skull in a case of carcinoma of the thyroid gland. The Roentgen findings were interesting, in that there was a radiating appearance of dense areas extending beyond the skull line into the soft parts of the scalp. In this respect the growth resembled that usually seen in sarcoma of bone, but microscopic examination proved the tumor to be of the same type of carcinoma as that in the thyroid.

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*See page 121.

Dr. Culbert said that it would be interesting to know if the carcinoma had extended beyond the temporal bone. Another point he would like to know about was the ultimate result. Dr. Eagleton had not entered into details; he had merely said that the wound healed kindly, but did not state what was the outcome.

Dr. Danziger said that two and a half years ago he had seen a case of carcinoma of the mastoid, though he did not make that diagnosis early, for it developed very insidiously. The patient had had an otitis two weeks before she was confined, but had paid no attention to it. After the confinement she sent for a physician, who had invited him to look at the case. There was no swelling, no temperature; on looking into the external ear one could see only a narrowing of the canal, which looked almost like a furuncle, and there was a little gland under the tip of the mastoid. The patient, however, complained of excruciating pain in the ear, and had much pain over the mastoid. On opening the mastoid the whole bone was found to be filled with thick tough granulations; the facial ridge was eaten away. It was a rather unusual picture. Some pieces were excised and sent to a colleague for examination, who reported the condition to be carcinoma. Before operation it was not possible to make a clinical diagnosis of carcinoma; it was not metastatic. The patient died.

Paper: Report of a Case of Gumma in Fossa of Rosenmüller, Causing Stenosis of the Eustachian Tube and Deafness.*

BY ISAAC M. HELLER, M. D.

DISCUSSION.

DR. GLOGAU asked Dr. Heller whether or not he found a predisposition, due to pathologic change within the nose and pharynx, such as described by Gerber and Gradenigo.

Paper: X-Ray Diagnosis of Perisinus and Epidural Abscess in Mastoiditis—Lantern Slides.

BY GEORGE S. DIXON, M. D.

DISCUSSION.

DR. LAW said that Dr. Dixon had covered the subject so fully there was nothing left to say. The plates show the condition absolutely as it exists, and demonstrate the value of X-ray in mastoid conditions. There are certain types in which the condition in the mastoid is worse than the symptoms indicate, and a properly made plate will help to clear up the diagnosis. Dr. Dixon had hardly emphasized sufficiently, however, the necessity for the roentgenologist to know the symptoms, the clinical history, and the pathologic findings. The X-ray is a very valuable help, but only a help, and the roentgenologist should be as familiar with the conditions as the physician. The best way is for him to have the history of the case when the picture is made, and then for him and the physician to study the plates together. There are many otologists who are capable of interpreting their own plates, and more who are not so proficient. There are conditions which cast somewhat similar shadows which are difficult to interpret and which require clinical and pathologic data to clear up. The otologist knows the data and the roentgenologist knows the significance of certain shadows, so a consultation between the two will aid materially in arriving at a definite conclusion.

The value of the X-ray had been demonstrated to a great extent in postoperative cases that do not clear up but continue for week after week. In many instances a roentgenograph will show cells in the mastoid which have not been cleared out.

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*See page 70.

Dr. LeWald complimented Dr. Dixon on his beautiful demonstration, and agreed with him thoroughly on the value placed upon the X-ray findings. If they agree with the clinical findings they clinch the diagnosis. In certain cases the Roentgen findings alone may be so distinct as to indicate that operation should be performed.

Dr. Guttman said that he would be glad if Dr. Dixon would give a little more of his technic in taking the pictures, and tell how he places the patient, etc.

Dr. Heller said that he also had read Gerber's article, but that in this case the nose was absolutely normal.

NEW YORK OTOLOGICAL SOCIETY.

Meeting of May, 1916.

Sinus Thrombosis.

Dr. A. B. Duel said he wanted to report briefly a case of sigmoid sinus and jugular thrombosis, presenting some unusual features, which he had recently operated. The case at first presented the usual early symptoms. At the end of the second week, when he saw it for the first time, all of the classical symptoms—repeated chills, widely vaccilating temperatures, bacteremia, etc.—were found.

On operating, he found a clot extending from the torcula to a point below the clavicle in the jugular. The jugular was tied through the clot, about an inch below the level of the clavicle. It was most difficult to dissect the vein out, owing to the chain of enlarged cervical glands, along its course. He was certain that the cord-like structure he tied off was the jugular only after he had found the facial vein containing blood, and followed it to the jugular by careful dissection.

It was most unusual in his experience to find a clot extending so far down the jugular—probably because one seldom operated now in the second week, the diagnosis being made much earlier than formerly.

The patient, after a very stormy period following the operation, was apparently recovering, after resorting to repeated blood transfusions.

DISCUSSION.

Dr. Rae said he would like to ask Dr. Duel what the blood culture had been after the operation.

Dr. Berens said he would like to ask Dr. Duel how far down the neck he had to go in order to find the clot, and if the vein was collapsed above the clavicle.

Dr. Duel said the vein was occluded by a clot well below the clavicle, and he was unable to tie below it. It was filled an inch below the lowest point he was able to tie off the lower end. As he pulled up the severed end the clot bulged from the interior of the vein.

Before he had cut it off, in order to be perfectly sure he had not gotten the nerve, he followed the facial vein down to the occluded jugular, which looked like a whip-cord and was about the size of the pneumogastric nerve. A streptococcus organism was found in the occluding clot, the vein wall and the glands.

Mastoiditis—Cholesteatoma.

Dr. W. C. Braislin said he wished to speak of a case, which to him was rather unusual, that he had seen during the last winter. He had never previously seen a case just like it. The patient had had a discharge for many years, since childhood, in both ears, and lately pain over the mastoids with a good deal of headache in both the temporal and occipital regions. The unusual feature of the case was that both meatuses and both canals were tightly packed with apparently a true cholesteatoma throughout. It had been partly removed by her private physician, who sent her to Dr. Braislin. It took him a good many sittings, eight or perhaps ten long sessions, to remove these masses. There also seemed to be some cholesteatoma inside the middle ear. All he could see he removed from a large perforation in either side. During the treatment he had anticipations of operating on the mastoid several times, for the patient continued to complain of severe head pains, and had not been able to sleep nights, except one or two hours together.

After clearing the thing up it rather promptly ceased discharging. When healed it was evident that pressure had existed for some time from these cholesteatomatous masses, from the present great amount of atrophy. The canal was very large after removal of the masses, and seemed to extend well up into the attic, although there was no connection between the canal and the attic.

Dr. Berens said he would like to ask Dr. Braislin how long this discharge had persisted before the sister came to him.

Dr. Braislin said that this woman had had the discharge for several years, and the trouble since childhood. He had not anticipated reporting the case or he would have looked it up.

Dr. Toeplitz said he would like to ask Dr. Braislin if this mass had filled the entire canal.

Dr. Braislin said the entire wall was filled up in an organized mass, which seemed to be in every respect a true cholesteatoma. The hearing was normal after the operation.

DR. QUINLAN said this case recalled to him, not in a microscopic way, but macroscopically, a case which he had operated on six years previously, of a growth from the external auditory canal, which had been present forty-nine years. Occasionally the mass would bleed, and the patient had been told by two or three men that the mass was possibly malignant and it had better be let alone.

The speaker said from the ordinary external examination he could not find the source of its attachment. There was a broken down area outside of its fibrous capsule. He made a postauricular incision and turned the auricle forward and found, close to the lower border of the drum and canal wall, a myxomatous mass with a broad pedicle. He had seen the patient recently, and after six years there had been no return. The growth was examined, and the speaker received a rather indefinite reply, stating that it was a sort of fibromatous mass. He did not know how fibrous tissue would cause such an appearance as this macroscopically.

Tuberculosis Following Mastoid Operation.

DR. CHARLES E. PERKINS said he had been impressed with the number of patients that came to the hospital or dispensary presenting no symptoms of tuberculosis, and after the operation had been performed and during the healing of the mastoid wound, they developed pulmonary symptoms and the examination showed pulmonary tuberculosis. Nothing had occurred previous to the operation to lead one to suspect that the lungs were involved, and yet during the healing of the wound they developed or were found to have tuberculosis. In the past four or five years the speaker had seen something like twenty cases with this history, and he had been wondering whether or not they were primary cases of tuberculosis in the mastoid process, or whether they had operated on pulmonary tuberculosis cases where the mastoid infection had been secondary. The speaker thought that it was often a hard matter to decide. He said he would like to hear the opinion of some of the fellows on this type of case.

DISCUSSION.

Dr. Toeplitz said he had a case in mind which had been occupying him for a year, and related to that question, and he would like to report the case, since it had always been interesting him as to whether the tuberculosis was in the mastoid or not. During the past thirty-one years he had seen a great deal of tuberculosis, and did not remember any cases of mastoid similar to this. It occurred in a child two years old, less two months, with a large mastoid abscess on the left side. There was also a swelling of the neck. He opened that mastoid abscess and evacuated an enormous amount of pus, which had quite an odor. The swelling on the neck was also opened, and he found an abscess which was not connected with the mastoid, but the entire structures were undermined as far down as the clavicle, and the pus with the odor, which he evacuated, amounted to nearly two cups full. The child was then treated for about six months, and the speaker battled with the case and finally succeeded in closing the wound of the neck, but the wound of the mastoid did not close. The child was then treated for the mastoid wound, being seen every other day at his office. The patient was extremely anemic, and Dr. Toeplitz had suspicions of tuberculosis. The parents of the child were also examined, but they did not show any tuberculosis. Finally, the child had a habit of playing in the mud, using mud as a plaything in preference to anything else, so that it came about that the dressing was frequently removed, and one day the speaker found an amount of maggots in that mastoid wound. He cleaned the mastoid out and despaired of closing it and turned the case over to a colleague for hospital treatment, who thought he would build the child up first, give it treatment and then try to close the mastoid wound. A few days previous to the meeting he had been told that the mastoid wound had not closed as yet, and was in all probability a case of tuberculosis of the mastoid.

That was one case, Dr. Toeplitz said, which came as near to the diagnosis of tuberculous mastoid as any he had ever seen, and it was now one year and two months since the operation and the opening was still there. He thought it an unusual case, and had so far been always able to close the mastoid, except in cases which he wanted to keep open himself, as in cholesteatoma, he had always closed the wound in these cases. This was the first case like it the speaker said he had seen in thirty-one years.

Dr. Rae said that there could be no doubt of the existence of a primary focus of tuberculosis in the middle ear, and that this was essentially true in the case of children. He briefly outlined a case of a baby about three yeares old, admitted in a stuporous condition to the Manhattan Eye, Ear and Throat Hospital during the winter. The child was an exceedingly well nourished baby. On examination the diagnosis of mastoid with tubercular meningitis was made. In spite of the meningitis it was decided to open the mastoid. Before doing so the laboratory was asked to provide the caustic soda solution so that pus directly from the antrum might be examined for tubercle bacillus. On removing the cortex a distinct pool of pus was exposed, and a specimen from this was withdrawn by a pipette and transferred to the soda solution. On examination this was found to be loaded with tubercle bacilli. An attempt was made to demonstrate the tract of infection to the meninges by removing the whole of the antral roof, but no such path of infection could be shown. The operation did not influence the course of the case, and the child died of tubercular meningitis.

Dr. McCullagh said he would like to ask if in this case of Dr. Rae's there had been any healing of the operative wound. It seemed possible that some of these cases might have been latent or unrecognized tuberculosis in which the ether caused a rapid development.

Dr. Quinlan said he would like to know if there had been any lymph nodes or broken down glands in the cases just reported by Dr. Perkins.

Dr. Perkins said the cases he spoke of were in adult patients, not the type Dr. Rae had been speaking of. Several of them he remembered in detail, who had no lymph nodes, but the patients looked pale and bad. After the operation he had had the lungs examined, and they were tuberculous. As a rule. they were very slow in healing, many remaining open when the patients passed from observation. Most of the wounds did better after the patients received the fresh outer air treatment, for it had not seemed right to keep them in the environment they were in, and they were, therefore, allowed to go to the country before the wounds were healed.

Dr. Quinlan asked whether these cases, of which Dr. Perkins spoke, were a tubercular infection of the mastoid—that is, having its characteristic features.

Dr. Perkins, in answer to Dr. Quinlan, said that he was not advancing any such theory of tuberculosis. As Dr. Rae said, it had been demonstrated that there could be a primary tuberculosis in the mastoid and middle ear. In children such tuberculosis was more common than was generally supposed.

Dr. Rae said his patient had no lymph nodes whatever.

Dr. Haskin said he would like to ask Dr. Rae if the cases he referred to were chronic cases. He asked Dr. Perkins if tuberculosis had at all been suspected from the appearance of the secretions in the canal, in the first place, in these patients.

Dr. Perkins said that no examination of the aural pus for tubercular bacilli had been made previous to operation, as tuberculosis had not been suspected.

Dr. Haskin said they had found tuberculosis constantly at the Manhattan Eye, Ear and Throat Hospital, probably twenty to twenty-five cases, within the last year. A boy patient had a profuse discharge and a great many tubercle bacilli were in the secretion. He had another patient, a school teacher, which Dr. Rae had also seen, in which the diagnosis was doubtful. There was a tubal catarrh, and they did not know what the thing was. He told Dr. Rae at first that it looked like a tubercular membrane, but the woman had no other evidence. On inflation the tube was perfectly dry, and yet there was a thickened membrane with a peculiar yellowish condition in two places. The speaker finally advised that a paracentesis be done, which they did, and since that she had a profuse secretion from the canal. The condition was absolutely painless. He said the Friday previous they took some secretion from the canal, and after putting this through a sodium solution and centrifuging it, they found tuberculosis. This woman had a tubercular inflammation of the drum membrane, and yet no evidence of a tubercular condition.

Dr. Haskin said that Dr. Lewis has reported three cases of tubercle bacilli at the last meeting, and Dr. Berens two or three cases with sequestra in the canal, and the condition was found all the time in the hospital. In the probable appearance of a lung condition after operation, there was the fact that the cases may have had it all the time, but they had enough vital resistance in the tissues to withstand actual breaking down. Then the shock of operation and loss of blood causes a tremendous lowering of the vitality, and the secondary infections were able to overcome the resistance of the body and the evidence of tubercular lungs was likely to appear.

Dr. Duel said he thought it quite probable that all of the gentlemen present had in past years operated on a number of cases of tuberculous middle ear and mastoid, being unaware at the time that they were dealing with tuberculous condition. He felt quite sure of this in his own case.

By the present methods of diagnosis a great many cases of tubercular middle ear or mastoid were discovered which tended to alter their view of former results. There were a great many cases of tubercular mastoid or middle ear that would eventually die of tubercular meningitis, but also a number of cases which recovered promptly after operation, without any such grave complications. When one thought of what the pathologists had said, that ninety or ninety-five per cent of the people coming to autopsy had suffered from tuberculosis some time in life, one might form a different prognosis in those cases in which they found tubercle bacilli present in the pus from a middle ear or mastoid abscess.

The speaker said he had operated on a case in an adult, which a number of men had seen on account of the pain, bearing out Dr. Perkins' idea, that not all tubercular processes were painless. The man had two badly broken down mastoids. They thought by draining them they might simply relieve his pain. The patient now has no discharge from either ear. Both mastoids were apparently entirely healed.

Dr. Braislin said he would like to ask the chairman if he would give them some of his ideas in regard to the treatment of the tubercular condition. He remembered two cases, apparently typical cases of tubercular conditions of the middle ear in adults, which began to suppurate without pain and had a rapidly melting down drum and other evidences of tubercular ear, one of which had been cured by X-ray with a very good result. He had another case of middle ear suppuration, which was cured by his removal for the winter to California. The meatus was large, and he was advised to allow the sun's rays to fall directly into the ear.

Dr. Quinlan said he thought that in children especially, the treatment with tuberculin had proven very satisfactory. He did not know just what the results were turning out in grown people, but he thought that tuberculin was a very valuable remedy, and also thought that in the past they had all started to use tuberculin too vigorously. They had paralyzed patients with too large doses of tuberculin, who were overwhelmed with bacilli. Dr. J. Solis-Cohen of Philadelphia had pointed out, two or three years previously, that in his work he was getting wonderful results by the use of tuberculin, and he used about 1/1,000 part of the dose which had been used. He reduced this down to 1/10,000, and gave five minims of that. After he had used up a certain quantity, he gave 1/5,000, and finally a dose of 1/1,000.

The speaker had one case in a clinic patient, a poor child, who had never been out of the city, which would not heal under any other form of treatment, but finally recovered under tuberculin. As far as local treatment went, he thought that one was about as good as another. The speaker thought, with Dr. Perkins, that the majority of patients, if they could be sent away to the country, with plenty of good air and sunlight, that it would be better than anything else that could be done for them.

Dr. Lutz said he knew of a case of a child, which had been treated with tuberculin, which was very much improved and practically cured. Another child, which had apparently a tubercular mastoid, was treated with tuberculin, beginning with very small doses. If he rightly recalled it, Dr. J. Solis-Cohen's article first appeared in the *Therapeutic News*. It had been of interest because of the small doses noted, such as 1/10,000 and 1/20,000, and the gradual way in which they were worked up from these infinitesimal doses, also interested the speaker. He had one little patient at the present time, who had a recurrence of her tubercular condition in the ear. There were some glands appearing, posterior to her mastoid, and Dr. Lutz had told her family physician to start her on tuberculin.

Dr. Berens said that at the Manhattan Eye, Ear and Throat

Hospital for the last year or two, instead of running these tremendous doses, equivalent to 50,000, he now came down to 100 and gradually ran it up. That winter he had had three cases of gland disease, two having been operated upon and one not. All three had done remarkably well. Two of them were well, so far as appearances were concerned, and the other promised to be well within two or three months, judging from the appearance of the glands.

Dr. Braislin said he would like to ask the gentlemen present about the method used in obtaining tubercle bacilli in the discharges.

Dr. Haskin said in regard to secretions, that at the Manhattan Eye, Ear and Throat Hospital they washed the canal with a sodium hydroxid solution, three per cent, which had a property of dissolving practically all other bacteria or cocci, except the acid fast staining bacteria. After that it was kept two or three hours in solution and then centrifuged and put on a smear and stained with the regular carbol fuchsin stain. In getting living cultures they syringed with the sodium solution, as before, and after thirty minutes centrifuged it and spread the deposit on tubes with Petroff's media. This media was discovered by Dr. Petroff, now at Saranac, and his original media had consisted in agar agar and beef juice; the speaker did not remember what else it contained, but thought a gentian violet stain. A gentian violet had been used, with the idea that this would kill off all the other bacteria in the growth and eventually the tubercle bacilli would appear in pure cultures, but the sodium hydroxid solution obviated the necessity of this. It was very pretty to see the growth occur in these tubes. Possibly in ten days to two weeks the pure colonies of tubercle bacilli appeared. They afterward did not use the gentian violet at all, and the cultures grew just the same. The preparation of Petroff's media had been given in various journals. It was very easy to make smears and have the cultures take place. Dr. Dwyer, who did the bacteriologic work at the Manhattan Eye, Ear and Throat Hospital, would give all the information about it.

Dr. Haskin said that if they washed out the ears with normal saline solution, this often failed; but with the sodium hydroxid solution they very frequently found the tubercle bacilli. Almost characteristic of all these cases was the profuse creamy discharge. There would be no pain in the old mastoid, and yet the profuse discharge kept on coming out of the ear. The speaker said one has to be very careful to differentiate the tubercle from the sirregma bacillus, which is also acid fast.

Unhealed Mastoid.

Dr. F. J. Quinlan said that three months previously they had admitted at St. Vincent's Hospital a case of fractured skull, in a truck driver who had been tossed from his wagon, and after remaining in the surgical division for two weeks had developed middle ear infection through the site of laceration of the canal. After a week or ten days the process went on to mastoiditis, and he had the characteristic symptoms which demanded operation. At the operation the speaker found the entire mastoid more or less broken down and filled with a creamy pus, and the sinus was exposed and looked rather suspicious, but on account of the bleeding that was associated with it, he thought it wise to let it go. About a week or ten days the house surgeon was disturbed one night with the news that this man had a profuse hemorrhage. When he came down he found the dressings filled with blood and the man almost exsanguinated. The condition was staunched and Dr. Quinlan saw the patient the next day. The day previously the temperature had been 105½°. He explored the cavity and examined the sinus. At the time of operation he uncovered as much of the bone covering the sinus as was necessary. At the exploration the next day he found the sinus looking badly, and was assured by the appearance that there was a parietal clot. He removed as much of the sinus as he could, in spite of the hemorrhage that preceded it, and was able to staunch both ends by wicks of gauze, low down near the jugular and as high up as he could towards the torcullar. The man went along fairly well, and the speaker said it seemed as if he had mastered the situation, but they noticed the wound was bathed in a profuse discharge. It was now six or seven weeks since operation, and there was absolutely no evidence of healing. The flaps looked like beefsteak that had been cooked, with no evidence of any attempt at healing. A Wassermann test had been made also, and several attempts to ascertain if it were a tubercular condition.

Even at the present time there was the same low grade inflammation. The man seemed fairly well nourished, but the wound would not heal and looked like a sluggish, inert, dead mass. The speaker had used balsam Peru, iodoform, everything therapy could suggest, and still there was the same condition. He had tried approximating the wound, local applications and general tonics, and the thing seemed almost like a labor without result. He said he would be glad to hear suggestions in regard to this wound—that is, stimulating it to better activity. He never saw a wound secrete more pus and show fewer granulations with less evidence of healing than this one.

Dr. Quinlan said this man was not more anemic than one would find in a man who had had more or less shock from a fracture of the skull and impoverished condition due to loss of blood. The man, he thought, had been alcoholic, but there was nothing upon that score to attract any attention. There was now no evidence of temperature, and the man ate fairly well and digested his food.

DISCUSSION.

Dr. Haskin said he would like to ask Dr. Quinlan the nature of this infection.

Dr. Quinlan said the nature of the infection was a pure streptococcus.

Dr. Haskin suggested the use of gentian violet, one to five thousand. It was used on the other side, where they found it most useful in dressing sloughing and gangrenous wounds, a one to ten thousand solution being even better than a hypochlorate of soda. He had noticed this in a journal of the military association. It had been used many times, and might be of value in stimulating this wound.

Dr. Duel asked if the ear was still functionating on that side.

Dr. Quinlan said the ear was still functionating somewhat on that side. He had tested the patient two weeks previously, and he could hear a voice about six feet on that side.

Dr. Toeplitz said he would like to ask if there were any hidden abscesses possible in this case.

Dr. Quinlan said the wound was so large that it had no recess, no fistulæ. He dressed that case daily and found no sinus, only this large, yellowish white area. He thought at first there was probably some sinus back towards the occiput, but there was absolutely nothing, and every part of the canal was free to inspection.

Dr. Berens said he would like to ask Dr. Quinlan if he had tried any vaccines in this case.

Dr. Quinlan said he had not tried vaccine, as his results with vaccine had not been satisfactory, and he thought he would not use it in this man, although everything had been done with local applications and constitutional remedies. In a day or so they might try something else. He was not altogether favorably drawn to the use of vaccine; still he said he would be glad to give the patient the benefit of any mode of therapy that would aid him toward convalescence.

Dr. Perkins said he would like to ask if the germs in the pus were still streptococcic.

Dr. Quinlan said that up to a few days previously the germs were still streptococcic.

Tonsillar and Mastoid Abscess.

Dr. S. H. Lutz said that on the Saturday previous he had seen a baby one and one-half years of age, which had interested him very much. There was a history of a running ear for a week or ten days, and a history also of sore throat for two or three days. It was stated that there had been something the matter with the baby six months before. The patient had a protruding ear and a prolapsed posterior superior canal wall. There were a large number of glands below and behind the mastoid. The speaker suggested operation. He looked at the baby's throat, and the tonsil on the same side as the ear protruded downward and inward, without any signs of tenderness or acute condition of the tonsil at all, but the location of the tonsil led him to put a knife into the anterior pillar and he got a large quantity of pus. With artery forceps, introduced to dilate the incision, he hit the anterior inferior surface of the mastoid. There was no question that this mastoid was perforated at the anterior inferior surface and the pus collected in the supratonsillar space. He had seen the same thing in a girl, which had been reported at one of the meetings of the society. Dr. Lutz said he had also had the same experience in a man about fifty years of age, who came to see him with an acute ear, and the speaker then lost track of the patient. When he again appeared he came with a history of bad sore throat, and there was a sinus in the upper part of the tonsillar space, which went directly into the mastoid bone. At operation later the speaker found the whole anterior surface and the tip broken down. The girl died, the man got well, and he did not know what would become of the baby, as he had not seen the baby again.

DISCUSSION.

DR. DUEL said this little girl, of whom Dr. Berens had spoken, had a suppurating sphenoidal sinus. However, the speaker said he remembered a case in an old man, where after a perforation of the mastoid tip the discharge had found its way into the peritonsillar space and there formed a large abscess.

Mastoid Abscess; Probable Meningitis Minus Temperature.

DR. S. H. LUTZ said that a week previously he had had a case of mastoid in a little girl, eight years of age. There was a history of illness before the operation. Both ears were involved, the left ear being worse. The speaker operated on that ear only, as the family were very much opposed to having the two ears done, and he operated on the left one with the hope that after the patient had been in the hospital a few days the family would be willing to have the other ear done. After the operation the child's temperature dropped, and at no time since had been above ninety-nine degrees, but for the past twenty-four hours there had been a great deal of headache and the child complained decidedly of the other ear. Examination of the eye grounds showed no signs of intracranial pressure. The child was carefully watched and the house surgeon reported that there was some stiffness of the neck and other signs of meningitis. There was no temperature, and on spinal puncture a perfectly clear, sterile fluid was obtained. The right mastoid was opened, and a peculiar condition was found. The cells along the zygoma and horizontally backward from the antrum and perpendicularly downward along the anterior border of the mastoid to and including the tip, were very much involved, but a clear space in the middle of the mastoid was not involved at all. The next day the child was more drowsy, but there were no eye symptoms, no headache, no temperature, and no localizing symptoms of a brain abscess. The case was presented for help, as it was unique in the speaker's experience.

DISCUSSION.

Dr. Berens said he recalled one case of meningitis particularly, in the hospital, where the temperature was almost entirely absent, in which there had been a thick, creamy cerebrospinal fluid. The patient had gone into coma and had died. The speaker said he reported this case two or three years previously.

Dr. Perkins said he would like to ask Dr. Berens what cell count there had been in the spinal fluid in his case.

Dr. Berens said, in answer to Dr. Perkins, that he thought the pneumococcus had been found in the spinal fluid, in the case to which he had referred without temperature.

Dr. Haskin said that regarding the first case which Dr. Lutz had mentioned, he had a little child at the hospital with a most unusual condition, one which he had never seen before. The patient was a boy, about ten years of age, and at the entrance of the external meatus and floor of the canal there was a hole about the size of the lead of a pencil. The speaker gradually dug out some cholesteatomatous masses and washed out the ear, and the day previously he put in a very fine probe and went down an inch and a half into this cavity. In probing towards the ear he could not detect any bone with the probe against the mastoid, which was a very peculiar thing. The boy several years ago had a glandular involvement with an extensive scar on the neck. The speaker said he had never seen a fistula like this, and wondered if it was an old Bezold perforation which had broken down and left a fistulous tract through the canal.

Mastoiditis Due to Teeth.

Dr. W. H. Haskin said that a man had been sent in to him from Freehold, New Jersey, four weeks previously, with a very violent inflammation of the middle ear and tenderness over the mastoid, and the speaker did a paracentesis. The patient ran a temperature with a streptococcus hemolyticus involvement, but in going over the man's mouth he found a contrivance on the teeth, which he presented to the society. He pulled out a tooth and it was attached to this bridge, and when the second molar came out he found it opened into the antrum. The speaker washed out the antrum through that canal and the stench therefrom nearly overcame the patient and himself. The speaker said this was a beautiful specimen of bridge work which the man had had in his mouth, and that after the removal the mastoid involvement rapidly cleared.

He showed another piece of dental work which had been taken out of a doctor's mouth. The bicuspid had been taken out under local anesthesia, and necrosis had produced a perforation in the alveolar process through which one could put a little finger. He said he would like to have some of the gentlemen look at the bridge work contrivances.

Another case he mentioned was one of the most serious he had ever seen, which had been referred to him about two weeks previously, suffering from some profound toxic anemia. He could not say that it was pernicious, because they had not been able to find any nucleated red cells. The woman first had hemoglobin, twenty per cent, 1,300,000 red cells, the white cell count was 4,200, the lymphocytes 8.0 per cent, and the polynuclears four per cent. A general medical man had examined her, also a surgeon, and they had found nothing to cause the trouble. Dr. Haskin was asked to go up to White Plains to see this patient. He examined the patient's mouth and took some fixtures from the teeth which had been fastened on the roots of twelve different teeth in her mouth, and every one of them had an abscess at its roots, which was shown by radiographic pictures. The woman had been suffering for years with a slow, gradual toxemia, with increasing lymphocytes and anemia coming on, without knowing it. She had no pain, and never suffered with pain, and was surprised when the speaker told her that her trouble was in the mouth. At first sight the right side of the jaw looked like a huge sarcoma, but on extracting the bridgework and pins, he found it was all broken down and the whole thing had since sloughed out. The roof of the mouth was typical of gangrene, but the alveolar process had not yet opened into the floor of the antra and nose, and he hoped it would not. There was slight evidence that this was going to form a line of demarcation, but he had had to cut out great masses of this necrotic tissue. It had been nothing else than a pure case of poisoning.

The speaker said that six weeks or two months previously he had been called to the Polyclinic Hospital to see a case in a child, who had been lying in the hospital for five weeks, white as a sheet, with the same condition. The hemoglobin count was fifteen per cent and the red cells about 1,100,000. The case had been examined by the general men and surgeons, who wondered what could be the matter, but no diagnosis was made. The pathologist asked him if the condition could come from the mouth. The speaker found necrosis of the right upper jaw from two badly diseased teeth. The whole upper jaw was sloughing out, and in the lower jaw there were two abscess cavities with profuse secretion and osteomyelitis. The child died two days later of nothing but a septic condition in the mouth, from neglected teeth, which nobody had looked at. The speaker said these cases were getting so common that he thought they ought to be constantly on the lookout and not trust the patients to the dentist. It might seem strange to hear, but he stated that not one dentist in ten thousand would recognize these conditions or would extract teeth in these septic cases. It had been recognized by a few men, who had been doing splendid work, and they had come to the conclusion that the dental profession has got to be jacked up. They could not blame the dentists, for their whole method of instruction consisted of gold crowns, fixtures, etc. The speaker said it was not safe to have any fixtures or such crowns, which were absolutely unsanitary and bound to create sepsis sooner or later.

DISCUSSION.

Dr. Lutz said he would like to ask why some dentists objected to pulling a tooth which was evidently covering an abscess. He stated that many dentists would balk at pulling a tooth that the surgeon would at once recognize had to be removed. Surgeons took teeth out and the people got well right away, and he wondered why the dentists were afraid to do it.

Dr. Haskin, in reply to Dr. Lutz, said that it was pure ignorance. The majority of dentists had no conception of the pathologic condition arising from these things. He said the only thing that could be done was, where one had a clean-cut abscess cavity and there was still a large area of sound alveolar process, that one could go in there through the alveolar process and amputate the roots at the apices and fill the canals, and the tooth would remain in a fairly useful condition for several years, but it was nevertheless a dead body which would gradually work its way out.

DR. QUINLAN said he would like to ask Dr. Haskin what they teach in the dental colleges, and why they did not teach such things as were mentioned by him, especially as the health and comfort of people depends so much upon the condition of their teeth. It is a source of pleasure to note that Columbia University has introduced a department of dentistry, and that the tocsin has been sounded throughout the country that attention to children's teeth is a great asset to their future well-being.

DR. HASKIN said, answering Dr. Quinlan's question, that these colleges were nothing but proprietary schools. The students paid a fee, and were taught to put on crowns and fixtures, and as far as scientific instruction went along the lines of pathology and bacteriology, they were absolutely unfit. They were crammed for the state regents and that was all.

DR. QUINLAN said he supposed they passed an examination in anatomy, physiology, materia medica, chemistry, etc., and that some great missionary should get up and teach them the errors of their past methods, and, if necessary, legislate against measures that sooner or later would bring about misery and incurable diseases.

DR. HASKIN said Columbia College had finally decided to have a branch of dentistry, but with six million people around here, and all the other big universities, like Bellevue, Cornell, etc., there was not one dental school that was alive to the existing conditions.

DR. QUINLAN said he thought there was a school of dentistry attached to Fordham University.

DR. HASKIN said he had heard of the universities of Harvard, Pennsylvania and California having dental schools, where the men had taken the first two years in medicine, which was little enough. The majority of dental schools were pretty bad.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of November 21, 1916.

THE PRESIDENT, DR. OTIS H. MACLAY, IN THE CHAIR.

Facial Deformity Resulting From Injections of Liquid Vaselin.

DR. GEORGE E. SHAMBAUGH showed the patient, together with photographs which the patient had taken immediately before and after the injections. The work was done several years ago by a "beauty specialist" in California. The victim thought his face too thin, and to overcome this, something over forty injections were made in the course of a day. Aside from a temporary congestion immediately following the work, the man was very much pleased with the results at first. It was not until a couple of years later that the relaxation and sagging took place which gives to the patient's expression the present ludicrous appearance. The sagging of the cheeks over the lower jaw on both sides is the most unnatural feature of the deformity.

The patient, who is a waiter, had expected to improve his station in life by making a specialty of just this kind of work, but the calamity which overtook the efforts on his own face fortunately kept him away from such work. He states that there is a great deal of this sort of work being done on the Pacific Coast, but that there is a good deal of it done here in Chicago. His case presents a fair example of what quackery does in medicine. The question is whether anything can be done now to relieve the condition.

DISCUSSION.

DR. NORVAL H. PIERCE thought that if hard paraffin had been used there might be a possibility of removing it to some extent. Of course, there was the chance that attempts to remove it might make the face even worse.

DR. WALTER S. BARNES said that someone has made a suggestion that heat might liquefy the contents of the injected material and permit it to be withdrawn with an aspirating needle.

Dr. Shambaugh thought that this applied more to the paraffin injections. His impression was that the lumpiness in the patient's face was more the result of connective tissue developments.

Leucoplakia of the Tongue.

Dr. George W. Boot presented a case of leucoplakia of the tongue in a man of about forty-two years, a peddler by occupation. Patient had had syphilis twelve years before, and had taken treatment for about three months. He had been a smoker, though not to excess. He has had no signs of syphilis since the three months of treatment. He presented himself for treatment because of a small painful spot on the side of the tongue. This had probably subsided under cleansing mouth washes, though the leucoplakia had been uninfluenced. The leucoplakia occupies chiefly the tip and borders of the tongue. It gives the patient no discomfort. Wassermann is reported as two plus. This case confirms the view that back of leucoplakia stands syphilis as the etiologic factor. This patient has been put on antisyphilitic medication, and it will be interesting to watch the effect on the leucoplakia.

Perichondritis of the Larynx.

Dr. George W. Boot presented a case of perichondritis of the larynx of unknown etiology. The patient is a butcher of about forty years of age. The trouble has existed for a year. He attributes it to catching cold going in and out of the ice box. He has had a tracheotomy done before coming under Dr. Boot's attention. The tracheotomy wound never completely healed. There remained a fistulous opening discharging pus. Later, swelling inside the larynx required reopening of the tracheotomy wound. When the tracheotomy tube was removed an up and down tube was inserted. After a couple of weeks this was removed. Patient did well for a time, when granulation tissue above the cords caused so much obstruction to respiration that it was removed by the indirect method. He now has plenty of room between the cords for respiration, but within the last two or three days a swelling has appeared between the epiglottis and the tongue on the left side. This has apparently opened and discharged pus.

In this case there is no suspicion of tuberculosis or malignancy.

Carcinoma of the Esophagus Rupturing Into the Aorta.

Dr. George W. Boot presented a specimen from an elderly man who had come to him for examination with the probable diagnosis of carcinoma of the esophagus. Dr. Boot passed the esophagoscope and found an ulcerating new growth near the middle of the esophagus. There was no bleeding from the examination. Two days later the patient had a sudden profuse hemorrhage and died. Postmortem examination showed an opening about one centimeter in diameter connecting the esophagus and the aorta. Only great gentleness in making the esophagoscopy could avoid hemorrhage at the time in such cases.

Carcinoma of the Epiglottis.

Dr. George W. Boot presented a specimen of carcinoma of the throat in which the growth appeared to have been primary in the epiglottis. The epiglottis was the size of a walnut. The new growth extended out into the right aryepiglottic fold and right pyriform fossa. The growth in the epiglottis blocked up the entrance of the larynx to such an extent that a tracheotomy was necessary on account of the valve-like action of the epiglottis.

The patient gave a history of having had antisyphilitic treatment for several months before coming under Dr. Boot's observation. In such a case as this early diagnosis of the nature of the growth and energetic surgical treatment should have made a cure possible.

Paper: The Tonsil Question in Children.*

By George W. Boot, M. D.

Paper: Clinical Problems Relating to the Faucial Tonsil in Adults.†

By George E. Shambaugh, M. D.

DISCUSSION.

Dr. Norval H. Pierce said it seemed to him that there was very little left to discuss. He heartily agreed with most of the things said in the two papers. He was happy to see that they condemned the massacre of the tonsils which had been going on. He thought that there was no doubt that there had been an enormous amount of unnecessary operations, especially in children. An adult can protect himself, but a child is the victim of his physician or overanxious parents. That is seen constantly at the dispensary of the Illinois Eye and Ear Infirmary and St. Luke's Hospital. He was sure he was right in saying that in the cases which he inspected immediately before operation, which had been prepared for operation, fully fifteen to twenty per cent were turned away and not operated upon. The visiting nurse and the physicians in charge of schools have been obsessed by the desire to remove all tonsillar tissue in the human race.

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*See page 129. †See page 135.

He considered the indications for tonsillectomies in the child to be about the same as those in the adult, with perhaps the exception that apparently in the minds of those in charge of children, all tonsils should be removed if they were at all enlarged. He agreed with Dr. Boot that the mere enlargement of a tonsil did not indicate operation. A tonsil can be quite large, but if it is not embedded and not the seat of recurrent attacks of inflammation, it should remain in the child.

As Dr. Boot had said, such tonsils very frequently undergo reduction in size very rapidly under syrup of iodid of iron or potassium. It is true that very little harm comes to a child from the removal of the tonsils other than the dangers necessarily dependent on the operation. It is remarkable how small a mortality there is in tonsillar operations, but he is quite sure that we do not accurately know the mortality. We accidentally hear about septic pneumonia, infarcts, hemorrhages, and sepsis, but very rarely read about them in the literature. He is quite sure that removing the tonsils in children must be a more or less dangerous operation; we know it is so in adults, and he believes we should be very circumspect in recommending any operation on the tonsil.

He thought Dr. Shambaugh had handled the question as to indications suffering from focal infections in a masterly manner. The crux in the matter in these adults he thought was represented in such a case as this: An internist sends a patient to a laryngologist for removal of the tonsils. He tells the specialist that he has very carefully gone over the patient with Bright's disease, or neuritis, or a rheumatic type of disease, and has found absolutely nothing; therefore, the tonsils must bear the brunt of suspicion. He recommends the removal of the tonsils; there is no manifestation of local disease; there is no history of sore throat, or only a slight one now and then, nothing can be squeezed out of the tonsils—no cheesy matter or pus. They may or may not be embedded. Should we yield to the internist and remove these tonsils? He admitted that he had been guilty of yielding in this way, but he has yet to see in such cases any benefit whatever from such an operation. It is probably true, although personally he had never seen such a case, that tonsils had been removed in cases with focal infection symptoms, in which the tonsils before operation exhibited no sign of inflammation, yet the operator has found at the base of the tonsil, absolutely sequestered, a small abscess. He has seen cheesy matter deep in the tonsil that could not be expressed by pressure, but he has never seen a real abscess deep in the tonsil without manifestation of it in the tonsil before operation.

He differed with Dr. Boot's remarks as to the depth of an anesthesia. He believes it is infinitely safer for the patient and very much more agreeable to the operator if the patient is carried down to a deep anesthesia so that the pharyngeal reflexes are abolished. The danger of the anesthesia largely rests in the up and down stages; if a patient can be kept down in one stage, there is no doubt that there is less danger from the anesthetic. Gagging causes bleeding, it throws an additional load on the heart, it interferes with dissection, and he believes it is necessary to thoroughly anesthetize the patient before beginning and then carrying that stage on until the operation is completed. Of course, speed in the operation is a desideratum, but safety and thoroughness are greater.

He is impressed by his experience in these cases that the cheesy deposits constitute one of the main causes for removal of the tonsils. If one consideration or another causes him to hesitate and he finds the cheesy deposits, that is the one thing that would cause him to advise removal of the tonsils.

He has yet to see a good singer hurt in any way by a tonsillectomy well performed. He has seen young women who said that their singing voices were entirely lost from removal of the tonsils, but he was in a position to know that they had never had a voice and never would have a voice. They were willing singers but not gifted singers.

DR. ELMER L. KENYON said that since the question of voice had been brought up, perhaps it was just as well to make a remark about it. Last winter he examined some forty or fifty tonsillectomized throats, with the purpose of determining exactly what had happened to the palatoglossus and palatopharyngeus muscles. In that series of cases five voices were found to have become permanently nasalized. That is a terrible percentage. If that ratio held in all operations, it would mean that one out of five had permanently nasalized voices; but of course that is impossible. These findings did not represent at all the true percentage, but they almost certainly did show that there was more injury to the speaking voice following tonsillectomy than any of us had thought. In all of these cases the trouble with the voice had resulted from a permanent injury to the palatopharyngeus muscle. That muscle had been pulled to one side and become adherent to the outer wall, with the effect that the soft palate was held down, making it impossible for it to move back to the posterior pharyngeal wall on phonation. Of course, one is likely to say that this happens only in badly performed operations. But these five cases were all done by different operators, some of whom he knew to be as capable of operating delicately on the tonsil as any operator anywhere; so it seems safe to say that it is not altogether bad operating that produces such results. It seems to him that such impairment to the palatopharyngeus may occur through infection following the operation as well as through injury to the palatopharyngeus muscle directly. If his hearers had studied the matter they would know that a very thin curtain of muscle remains to the posterior pillar after the removal of the tonsil, thus enabling slight injury to bring about a serious result. If no serious injury has occurred to the palatopharyngeus muscle from tonsillectomy, it may be that the voice can be handled very nearly as well following the operation as before. One has to bear in mind, however, that the tenseness of the neighborhood structures following operation is apt to be decided, and it was very difficult for him to understand how a singer could handle a tense throat as well as a relaxed one.

It was perfectly evident, and had been shown by the speakers, that one great difficulty was to determine when we should or should not operate. If there was no clear reason for operating, it seemed to be a serious matter to do a tonsillectomy. It seemed to him that the need was to develop a conservative operation which would fit doubtful cases and cases where the voice ought to have especial consideration. He believed that it had never been shown that an intracapsular operation was not capable of being successful if it were a thorough operation. He believed that future development in this field lies in the direction of finding two operations: one, the operation which we do, and the other a more conservative operation, which runs no danger of death from hemorrhage, and no danger of serious impairment to the contiguous important muscular structures of the throat.

Dr. Louis Ostrom (Rock Island, Illinois) said he would like to relieve his mind and also to present a grievance. We have had several men such as that prince of modifiers, Dr. Pynchon, in this socity, who have modified instruments, but he did not know that anyone had ever taken out a patent on a modification. Among these men are Drs. Beck, Freer, Ballenger, Boetcher, Tydings, Pierce, Ingals, etc. He had wanted a modification of the Sluder instrument made, but he was told that he could not get it because the idea of the dull blade had recently been patented. Several years ago, shortly after Sluder's first paper, Dr. Ballenger devised such an instrument, with one very dull blade and one sharp blade, the one to crush the base of the tonsil, the other to cut it off. Dr. Beck afterwards devised his snare for the same purpose. Last year at the American Medical Association meeting in Detroit, Dr. Makuen demonstrated a dull bladed Sluder guillotine, with which he uses a snare to slowly cut off the tonsil. Dr. Ostrom has had very bad hemorrhages in tonsillectomy by every method, and in cases where the tonsils were removed as clean and complete as they could be; but because he liked the Sluder method best, he made his original Sluder duller and duller, with the idea of crushing the blood vessels until he finally got it to about one-sixteenth of an inch; then finally he got to a point where, after leaving the tonsil in the instrument for some time (ten minutes), he would cut it off, but it did not work satisfactorily, so he modified Sauer's idea by adding a stop to the forward movement of the blade, so that when the blade was tightened with the screw it did not break the distal ring of the instrument. After a wait of several months he finally got a modified Sluder-Sauer guillotine made, but could not get another because the instrument maker told him that Ballenger's original idea of the double bladed instrument with one very dull blade had recently been patented, and his idea of a dull blade in the Sluder instrument was an infringement of this patent. He has been using the instrument (modified Sluder-Sauer, which he demonstrated) for about two months, and has had no hemorrhages at all if the tonsillotome is retained for a sufficient length of time. Blood will not coagulate in much less than five or six minutes at best in the laboratory, or sometimes eight to ten minutes, and it is not reasonable to suppose that you can produce coagulation in the blood vessel ends in all cases by putting on a snare wire and cutting off the tonsil in less than eight to ten minutes. By using two instruments at the same time, both tonsils can be removed in two or three minutes, and the instruments left in place as long as you like. During this time the adenoids can be removed. If more instruments are at hand, several cases can be attended to in a very short time. The tonsil is simply shaved off the flat side of the guillotine with an ordinary scalpel.

His protest is that it is not, in his judgment, fair that anyone now should patent Ballenger's original idea without giving him any credit, and especially when the idea is only one of a dull crushing blade which in itself is as old as the hills. He is now able to have his modified Sluder-Sauer made, but it took a fight with the instrument maker. Another member of this society, who is also present at this meeting, tried to get one made exactly like the one Dr. Ostrom demonstrated, but was refused because the dull blade idea was recently patented.

Dr. Ostrom had another little idea which is a modification of the Cushing-Crowe silver clip. By putting a little wire in here (demonstrating) to control the hemorrhage, no other artery forceps is needed. If you have a bleeding point anywhere, you use the tongue depressor to find that bleeding point, then, without using an artery forceps of any kind, you catch it and the little clip stays. He has used many of them, and they have never caused any trouble. He considered it the nicest little device for controlling hemorrhage that he knew of.

Dr. Henry F. Helmholz had come to the meeting to get the opinions of the men as to tonsillectomies in infancy and childhood, and he thought most of the men will find that the pediatricians, as a rule, are conservative in their recommendation of tonsillectomy. He thought the difficulty has come rather from the side of the parents. Parents frequently bring in their children with somewhat enlarged tonsils and ask if they shall be removed. If the child is in good physical condition and the tonsils show nothing abnormal, it is not difficult to give advice. Where, however, the child is below par, where the tonsils look suspicious, it is difficult to say more than that the removal of the tonsils may be of some benefit to the child. The question then can be put up to the specialist as to whether or not these tonsils will be removed. He finds that his cases group themselves into cases where he absolutely recommends that the tonsils must come out, and those cases where it is difficult to make any decision.

There is the question of the removal of the tonsils within the first two years of life. He has had a number of cases where a child had to be sent to a specialist a second time because the first time the specialist said the child was too young to have them taken out. One should consider that the younger the child the more likely the lymphoid tissue is to develop; but in instances of follicular tonsillitis there has been a tremendous improvement in the general condition of the child after removal of the tonsils.

Dr. C. H. Long said that Dr. Boot referred to the time of removal of the tonsils with enlarged cervical glands which were probably due to tuberculosis without any lung focus. It has been his habit to remove these tonsils and give tuberculin afterward. Last May Dr. O. W. McMichael, of the Municipal Tuberculosis Sanitarium, stated to him that he gave tuberculin first and afterwards removed the tonsils. He would like to know if that is the custom.

In February, 1916, he did a tonsillectomy upon a teacher who had exceedingly large cervical glands which had been present since the age of seven years. He expected to use tuberculin during the summer vacation, but the glands disappeared without it. The tuberculin test had been strongly positive.

Dr. Shambaugh spoke of the internist who referred the case to the specialist for operation. He would like to add one more specialist—the dentist. When adults are referred to Dr. Long by the internist, and there is any question about the tonsil being the source of the focal infection, he has the dentist exclude the teeth as a possible factor. The tonsils are then only removed when there can be no other cause discovered. The X-ray frequently indicates a root abscess when the patient has had no complaint of the teeth.

A young lady with a chronic sinus trouble, especially of the antrum, consulted him several months ago. He advised having a radiograph made, and saw no more of her until about a month ago, when she returned and consulted him about her eyes. She had consulted another specialist, and he had drained the antrum through the nose. The hole in the lateral wall had closed and the sinus was still discharging through the normal opening. He insisted upon a radiograph, which showed that the antrum was affected from the first molar tooth. She was referred to a dentist, who removed the tooth and the abscess cleared up promptly.

Dr. H. I. Lillie was much interested in Dr. Boot's assertion that the upright position in tonsil operations should be condemned. His training had all been in the upright position under ether, doing a knife and scissors operation. The anesthetic is given by the closed cone method, in the recumbent position, and the patient is put to sleep without much discomfort. He has had occasion to give many anesthetics, and to give them to doctors who believed, after the first few drops, that they were receiving chloroform. They never push the anesthesia beyond the pharyngeal reflex, and the patients are then put in the upright position. He considers it most important that the head must be held securely, as it is apt to drop. The advantages of this position are numerous: you see the tonsils in the same relation as when you examine the throat, the bleeding does not seem to be as great as in the recumbent position, the tongue does not interfere so much, and the bleeding is easily controlled.

In reviewing the literature last year he found a report of nine cases of lung abscess in which the authors concluded that the anesthesia was too deep, so that the blood went down the throat. In a series of cases in the upright position, it can be shown that there is no more blood in the larynx in this position than in the recumbent position. It is the contention of many men that the upright position should be condemned because the blood goes into the larynx, but as a matter of fact the blood goes down the pyriform fossæ and esophagus more easily than it does into the larynx.

Dr. Lillie believed that the stay of the patient in the hospital after a tonsillectomy is insufficient. Ordinarily twenty-four hours is the time recommended. Lung complications and septic complications do not show up in that length of time, and as most of the operations are done in the dispensaries or hospitals, these patients do not report back. He believed the time will come when the stay of a patient in the hospital will be increased to three days at least. In that time a lung infection or embolus will show up. In one case he had seen a lung embolus which no one could say was due to the upright position.

Regarding the question of pathology, most of their cases were in children, and they were fortunate enough to have a pathologic examination in every operated case, and it was surprising to note the percentage of tuberculous tonsils. The pathologist went over all the cases himself, and discovered that seven per cent of all operated cases in a series of nearly forty-seven hundred showed tubercular foci in the tonsils, the giant cell infiltration with occasionally tubercle bacilli.

Dr. Alfred Lewy said that in a previous paper Dr. Shambaugh had called attention to tonsillar infection and lesions of the eighth nerve. He would like to ask under what conditions in cases of nerve deafness he would advocate removal of the tonsils.

Dr. George E. Baxter said he had come with the idea of learning something about the attitude of specialists in nose and throat work in connection with tonsillectomies in children. He had been particularly pleased with the general sentiment of conservatism which seemed to pervade the meeting. He was also convinced that the last word in tonsil work had not been said. Discussions as to the methods of operating had been going on for years, and would probably continue. There is a tremendous field for observations and conclusions as to the morbidity in children following an operation, and a field which naturally belongs to the pediatrician. Few specialists are in a position to follow their cases and learn the ultimate results of surgery of the tonsils. Not until a large group of cases, studied over a period of years, are observed can we arrive at proper indication for and value of tonsillectomy in the child. He felt also that a warning which had been mentioned should be emphasized, against the operations for removal of tonsils in infant life. Dr. Helmholz had mentioned four cases where the indication for operation was definite—such indication must be most definite, and restricted in cases during the first two years.

Dr. Baxter felt also that too little cognizance had been taken of the postnasal tonsils. It is certain that large numbers of infections in the nasal and postnasal spaces, where the adenoid tissue is located, occur in the first two years of life. It is very common to come across children with running ears. These are not recognized as cases of otitis media until after there has been some external evidence. He was not yet convinced as to what was the best thing to do in these infants who have frequent severe infections in the postnasal spaces which frequently result in otitis media and cervical adenitis. Mention of the postnasal tonsil is made because of the close relationship with the faucial tonsils, and because adenoids are always operated at the time of the faucial tonsillectomy. This will admit of very much more study before coming to a definite understanding as to just what is best to do about tonsils and adenoids in young children and infants. Those cases for a long time have been sent to specialists without any particular recommendation other than the fact that the tonsils and adenoids are enlarged. His judgment was that we must come to some definite and concrete indication for operative work on the tonsils in children and infants.

Dr. W. A. Mann said he had been listening with a great deal of interest to the discussion of the papers, and he agreed with most of the statements. However, he felt that the statement that a large number of tonsils are removed unnecessarily should be confined to the general practitioner or surgeon who is not a throat specialist. He thought the specialists were very careful. His experience is that patients are sent by general practitioners for operation, but if there is no sign of disease he advises the patient to wait. Yet in a lot of these cases there has been evidence of infection within two or three years, so where there is any evidence at all he thinks it is a good thing, as a matter of prevention, to take the tonsils out. If the operation is properly done, it will do no harm and may do a lot of good.

Dr. Arthur R. Elliott said that in common with other internists he felt a great interest in the tonsil problem. He had no opinion to express as to the removal of the tonsils in children, as that was not within his scope. He could only express a personal conclusion in the matter of tonsil operations in adults. As the result of such personal experience as he had had he had come to several different conclusions to serve as a working basis. The first principle which outlined itself to his mind very definitely was one regarding the removal of tonsils in cardiac disease, using that term as being more conclusive than endocarditis, although endocarditis and pericarditis would include the majority of cases he had in mind. He thought that the tonsils should be removed where they appeared to be pathologic, or in all individuals who have valvular disease, irrespective of whether the endocarditis can be traced to the preceding tonsillar infection or not. The ground for such a statement would appear perhaps upon consideration of the fact that the tonsil and other lymphoid tissue comprises the portal of entry of the streptococci infection, since we know how much it is to be feared. Throat infections in individuals who have, especially in their valves, a point of low resistance are always a source of danger. Cases of malignant endocarditis, and the extremely virulent type of endocarditis known as subacute bacterial endocarditis, occur only in individuals who have had previous endocardial infection, and he believed it a thoroughly good practice to advise removal of the tonsils in all individuals who have valvular disease, as a prophylactic measure.

Regarding the removal of the tonsils in chronic internal disease, such as arthritis, nephritis, diabetes, etc., we have often to include the tonsil under the doctrine of the importance of focal infection which has been so prominent in recent medical thought. As Dr. Pierce had stated, the internist is often put to the last resort of considering the tonsil as the possible criminal in cases of this sort, and resorts to the process of "passing the buck" to the laryngologist. He entirely agreed with Dr. Pierce, that where the tonsils had been removed simply as a last resort in seeking for some escape from a difficult situation, without there being some obvious reason other than to serve an end, no good was accomplished. The removal of tonsils from which can be expressed purulent or seropurulent material, or tonsils which are the seat of recurrent infections, is of the greatest benefit and results in much improvement.

As to the depth of the anesthesia, he was sure it was of great importance. Nature has established a guard in the throat in the laryngeal reflex, which keeps us from inhaling foreign material, and if we abolish this reflex during our operations we open up the way for inhalation of infectious material. He was sure that lung abscesses are caused by the inhalation of such material.

He thought Dr. Kenyon was right. No matter how carefully the operation is done, there is is bound to be a considerable scar; and if the palatopharyngeus msucle is caught in this scar, there is bound to be damage.

He had the pleasure of seeing Dr. Ostrom operate by the use of his new instruments, and there was not so much as a drop of blood lost. His chief objection to the method is that if the instruments are used and left on for fifteen minutes, more anesthetic is required than would otherwise be used.

If all men who refer patients to the throat specialists were as careful as Dr. Helmholz, we would have no quarrel with them about who should be operated upon.

As to the method of anesthesia: at the Children's Memorial Hospital they use an ordinary paper cone with a towel around it and a little gauze inside. This is the method he prefers. Dr. Lillie uses a metal cone which is very similar. He does not like ether given for tonsillectomy by the drop method. The anesthetic is of great importance, and the more rapidly the patient is put to sleep the less anesthetic is required and the less time the ether has to become fixed in the tissues.

Dr. Shambaugh, in closing, said that the discussions had touched very little upon questions presented in his paper. The problem for the throat specialists is to decide whether the tonsils are infected in cases of systemic infection. This can often be determined from the history—as, for example, where there has been an acute tonsillitis complicated by rheumatism, endocarditis, or Bright's disease. In most of the cases, however, the onset of the systemic trouble is not associated definitely with acute tonsillitis. He stated that he had seen a patient this morning whose tonsils he had removed three years ago because of persistent neuritis of the brachial nerves on both sides. The trouble began in childhood, and the woman was now forty-five. The suffering had been much worse in recent years. During this period she had had no tonsillitis, but was subject to it in early childhood. The tonsils showed no superficial evidence of infection, but after removal, a number of small abscesses were found in the depths of the tonsil, containing streptococccus viridens. Within six weeks after the removal of the tonsils the neuritis disappeared and she has had no return. He recalled another case on which he had operated several years ago because of persistent neuritis of the right arm, of ten years' duration. The patient had never had tonsillitis or sore throat, and no evidences of infection could be detected about the tonsil; not even the cheesy plugs in the crypts were discovered. The operation was performed on the advice of the internist, because no other focus was detected. While operating a small abscess in the left tonsil was opened. The patient has made a complete recovery and has had no return of the neuritis. These are the most difficult cases to handle: where the tonsils show no evidences of persistent infection, and where there is nothing in the history that would throw suspicion upon the tonsils.

As to a relationship between nasal infection and tonsillitis: it is entirely possible that a chronic accessory sinus infection may constitute the carrier of infection, which could cause recurring attacks of acute tonsillitis. It is much more frequent, however, to find the reverse true. Patients suffer from recurring infectious head colds because of the persistent infection in the faucial tonsils. The removal of the infected tonsils often puts a stop to this tendency.

Dr. Shambaugh was asked to say something about indications for tonsil operation in cases of neuritis of the eighth nerve. In reply he stated that a degenerative neuritis of the eighth nerve is responsible for a great many cases of nerve deafness, and especially for those cases where attacks of vertigo occur. Formerly, these attacks were supposed to be due to hemorrhage into the labyrinth, or to a condition of angioneurosis. Neuritis of the eighth nerve occurs as a sequel of the acute infectious fevers. Occasionally it results from drug poisoning, tobacco or alcohol, but very often there is no apparent cause, and it is the opinion of the speaker that in these latter cases focal infection may frequently be the cause of the degenerative process in the eighth nerve, producing nerve deafness and tinnitus aurium, punctuated often by attacks of acute exacerbation, during which vertigo will be a symptom, provided the vestibular nerve is involved. In cases of chronic progressive nerve deafness, where a degenerative neuritis of the eighth nerve seems to be the cause, the case should be examined for possible foci of infection. If the tonsils are found distinctly infected, we should be guided as to their removal exactly as in cases of neuritis involving other nerves.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of December 19, 1916.

DR. OTIS H. MACLAY, THE PRESIDENT, IN THE CHAIR.

Case of Falsetto Voice.

DR. ELMER L. KENYON presented a boy of nineteen years with a falsetto voice which had been present since the age of fourteen.

Paper: Salient Facts Regarding Tonsils in Children and Adults.*

BY JOSEPH C. BECK, M. D.

(A reply to the papers and discussion of the last meeting.)

DISCUSSION.

DR. NORVAL H. PIERCE said he felt greatly relieved at the mildness of Dr. Beck's remarks. He had heard that Dr. Beck had been so incensed by something in the papers and discussion at the last meeting that he had feared to voice his displeasure at the time, and so allowed himself a month in which to quiet down. As a matter of fact, he did not see where he had replied to anything or done anything beyond giving his own views regarding certain matters, which are admirable. Still, his paper did bring out points for discussion. The axiom which he voiced at the beginning of his paper, "that a tonsil is better out than in," Dr. Pierce disagreed with. If we accepted such a position, it would eliminate all intelligent consideration of the subject. It would be very easy, if a patient came to you with a pair of tonsils, without any further consideration, to say, "Your tonsils must be removed." That mode of action has brought numerous procedures into disrepute. It is a position which surgical men have condemned in all branches, and it is the very turning point between humanely directed effort in medical practice and the veriest quack, who has nothing in view but his fees. There is something besides merely removing the tonsils; there is the danger that the patient is exposed to—a danger which has not been properly appreciated. The danger from the anesthetic and of shock, whether local or general; the danger of sepsis, the danger of pneumonia, etc.; and every patient who is subjected to tonsillectomy is liable to all these dangers. But aside from that, Dr. Pierce still claimed that the tonsil must perform some function in the human economy. This is shown by the fact Dr. Beck brought out—that even after a very careful, complete tonsillectomy there will be most pronounced hyperplasia. Nature is trying to do something to take the place of the excised tonsillar tissue.

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*See page 149.

As to age, Dr. Pierce was somewhat changed in his ideas regarding age being a counter indication to the removal of tonsils. In the last six months he had, in three cases, removed tonsils from children varying in age from two to three and a half years. They were children who had large tonsils, with repeated attacks of inflammation, who were not doing well, and the physician who had them in charge rather insisted that the tonsils be removed, and he removed them—with the most gratifying results. So he had decided that perhaps the age of the patient did not matter, if there is the indication for removal; but the indication for removal should always be present. The patient with tonsils is better off with the tonsils, Dr. Pierce believed, than without them, if the tonsils are not diseased.

Dr. Beck had touched upon the painful sore throat that followed tonsillectomies. The speaker had had some cases in which this had been a source of great annoyance, both to him and to the patient. He admitted that they had occurred in neurasthenics, men and women of pronounced hyperesthetic constitutions, but their sufferings had been real, and he had done everything that he could to relieve them, and yet the pain in the throat, which was not referred especially to the tonsillar region, but rather below, had persisted. He had explained this in his mind by supposing that the nerves in contact with the tonsillar capsules had been caught in scar tissue, thus causing a radiating pain low down in the throat.

Dr. Beck had spoken of the depth of anesthesia. With the insight of an artist he had said that he operates in a stage that is neither deep nor superficial, in which the laryngeal reflex is retained. That is the point of digression between Dr. Pierce's views and his. The operation is infinitely more difficult when the patient is retching than when the reflex is abolished. The hemorrhage is greater, the accuracy of the incision very greatly interfered with; and he reiterated his view that it is safer for the patient, and much more agreeable for the operator, if the laryngeal reflex is abolished.

As to the connection between otosclerosis and the tonsils, Dr. Pirce considered that purely hypothetic, and would not discuss it pro or con; he would only say with the utmost earnestness that it is purely a dream. There is no one fact that can be brought out to prove that there is a connection between the tonsils and the otosclerosis.

As to the connection between the tonsils and poliomyelitis, he believed that possibly we are on the threshold of very interesting disclosures. Many experiments have been performed, but Dr. Pierce thought they were still in such a primitive stage that they should hardly be brought out before such a discussion as this.

Dr. Arthur M. Corwin said he did not hear the papers and the discussion at the last meeting, but whatever their import, the result was good in stimulating Dr. Beck's present effort in a paper at once comprehensive and rich in the experience of the essayist. He believed the essayist eminently sane, and also Dr. Pierce, in most respects. He did not know that this discussion was needed in the laryngological society. He believed every man in the room was so thoroughly posted upon the tonsil from many angles, that there could not be wide disagreement among them regarding the reasons for a thorough tonsillectomy. When a patient comes into his office, and in the routine way he looks over that patient from bald spot to toe nails, as a laryngologist should, and becomes thoroughly acquainted with that patient—not through a narrow slit, but broadly viewed as an entity—he knows whether he ought to advise the removal of those tonsils or not, whatever the age of the patient. Seventy years or eighteen months was not a bar, in the presence of weighty indication for bettering the patient, reasons local, regional and systemic.

As to the minor differences: in a great subject like this, the difference in technic in handling hemorrhage, the procedure in operating the patient, the kind of anesthesia, those things will vary as widely as the shape of the noses in this room. While we each have our favorite method, we none of us are infallible. However good a man thinks his technic—however good his average results—if he will go and see others operate, it will do him more good than the reading of volumes or listening to hours of discussion. He did not wish to prolong the talk or bring out his own hobbies for exhibition purposes. But he differed in the way of controlling hemorrhage; never did he sew pillars together, never yet in many thousands of cases, old and young, did he have to ligate an artery in the throat. No matter how much one talked, one fellow would go on ligating and another fellow handle it in a different way. The thing is to take a tonsil out in the technically best and most complete way we know how—for the right kind of a fee.

Dr. Elmer L. Kenyon said it seemed to him that one could not talk in a final manner upon any subject where the knowledge was not yet complete, and certainly it was not complete concerning what we did, or might do, to the structures of the tonsil outside the capsule. He knew, for instance, that the palatoglossus muscle was in more than fifty per cent of the operations rendered partly or wholly incompetent, but he did not know of how much importance this muscle was to the speech. He believed that he knew that the socalled capsule of the tonsil could not be operated with any accuracy from the intrapharyngeal aponeurosis, and he thought he knew that in most cases the integrity of the palatoglossus was dependent upon the retention of at least the intrapharyngeal aponeurosis, and often the integrity of the palatopharyngeus as well. He knew from personal knowledge that the palatopharyngeus muscle had been in certain cases so badly destroyed, and the resulting scar tissue so harmful to the soft palate, as to render the patient's voice permanently impaired, even though the operation had been done by very skillful hands. From the standpoint of the singing voice, nobody knows just the value of the palate muscles in the production of the singing voice, but in all probability the palatopharyngeus especially is of importance.

Dr. George W. Boot believed that most of what Dr. Pierce and Dr. Beck had said was right, but he must disagree with part of Dr. Beck's statements. He had recently attended a meeting where one of the doctors urged that the people get the "tonsil habit." He thought it a bad habit for the people to get. It is bad enough for the doctors to get the habit. One of the first things impressed on him as a student of medicine was to do his patient no harm, even though he did him no good. He thought no one could deny that in removing tonsils the patient was subjected to some danger. Occasionally lung abscesses have followed tonsillectomy, and there are all manner of other bad effects that are seen, such as absence of the uvula, soft palate, artificial cleft palate, and so on; so that we can hardly deny the danger of the operation, and we have no right to subject our patients to such danger without justification. These results are not always from the work of beginners, but some of them have followed the work of very good men, and he thought one should be very careful in urging the laity to get the "tonsil habit."

Dr. P. J. H. Farrell said that Dr. Pierce had referred to the question of the anesthetic; he thought it was a well known fact that a complete anesthesia was the one which gave the least shock and danger; that applied to the throat as well as elsewhere.

In referring to what Dr. Boot said, the implication was to have school children examined thoroughly, including an examination of the eyes, ears, nose and throat. It was simply a matter of a thorough examination. He is chairman of that committee. The tonsil is the least important, as regarded from the standpoint of school children. Dr. Farrell had heard only the last few paragraphs of Dr. Beck's paper; he thought that no case of neuritis was ever cured by operating on the tonsils. Every case of neuritis was self limited, and just as apt to clear up today as tomorrow, whether there was an operation or no operation.

Another point which Dr. Kenyon mentioned was the effect of a tonsillectomy on the voice. Dr. Farrell was sure that no adult voice has ever been improved by a tonsil operation, and that many thousands of valuable voices have been ruined by operations upon the tonsils. Some patients will say that their voice is better, but they had no voice of value, either before or after operation. Many voices that return large incomes are ruined, their commercial value destroyed, by operations upon the tonsil; and the speaker thought one should be very conservative about operating upon the tonsils of professional singers or speakers. Dr. Farrell said most of the members present knew that he spoke from a very intimate knowledge of both sides of this question. In examining the throats of singers we find that the great majority of them have large tonsils, and the greater the amount of power the larger is the tonsil and the greater the resonance and vibration. If the palatopharyngeus muscle is destroyed, the case is hopeless, so far as restoring the voice of the singer or public speaker is concerned.

Dr. Robert Sonnenschein said he would take the liberty of speaking in regard to Dr. Corwin's remarks. He had followed his suggestion about seeing other men operate and had learned many things. He had seen Dr. Corwin operate by the Sluder method, and he had used the same method, but he could not understand how it was that he had never had to ligate a vessel. With the most careful technic, Dr. Sonnenschein had found it impossible to avoid ligation in some cases.

Dr. Arthur M. Corwin, replying to Dr. Sonnenschein, stated that the ordinary technic which he followed for control of hemorrhage was to operate the patient standing at the patient's right. As soon as he removed the patient's left tonsil, his assistant on the left of the patient went in with his finger wrapped with gauze, instantly controlling all hemorrhage. He then reached for the Corwin tonsil hemostat, which was fitted with a good sized pad that would fill the sinus, being larger or smaller, according to the case. That was put in immediately, carrying a little styptic—twenty-five per cent of tincture of iron in glycerin. He then went after the other, the near tonsil, in the same manner. If he was operating for adenoids, he went around on the other side of the patient and got the whole thing out in five lobes, with one application of the Stubbs curette manipulation, with a side to side and downward motion and a quick pull at the end. With the patient's head held so that the mouth was below the operation field, all bleeding into the larynx was prevented, and the adenoid invariably dropped out of the mouth as it was cut out with the last motion of the curette.

Dr. Corwin said the hemorrhage could be controlled by these hemostats, finger and sponge carrier, by a little judicious pressure; sometimes it takes a little more or sometimes a little less, but before the patient leaves the operating room, and before the operator leaves the hospital, he always sees that the hemorrhage has ceased. He has never had to ligate the vessels or sew the pillars together. He did not wish to say that those things were not right to do semioccasionally; but thought them inadvisable in routine. He thought a dogmatic attitude for or against measures that proved satisfactory to others and gave good results, was something a free-minded man should not have toward the other fellow, if he did not want the other fellow to bark and growl dogmatically at him.

Dr. Beck, in closing the discussion, said that if he had given the impression of operating every patient he saw, he wished to correct it. Tonsils were only taken out of patients when he thought they needed to have them taken out. He said his broad statement that everybody's tonsils would be better out than in was made because we do not know the function of the tonsil, except to know that it causes a lot of trouble. We believe it is a cause of disease—it is a nidus for infection, except for the first few years, when there is a hyperplasia. He believed that early in life such tonsils had something to do with the thyroid and thymus, and therefore he did not believe in removing them at that time.

So far as the dangers are concerned, they are not at all alarming, if you know how to handle the patient, in the opinion of Dr. Beck. He said that if his paper was going out as a "get the tonsil habit" paper, he was not going to publish it. He certainly did not agree with any tonsil habit or anything of the kind. Every point which has been made about the danger of hemorrhage, etc., he forestalled by strictly surgical measures. No matter whether the hemorrhage was stopped by pressure or by ligation at the time of operation, when the patient was returned to bed and began to use the muscles, fresh bleeding occurred, and the patient inhaled or swallowed the blood; and Dr. Beck thought if many of the abscesses that are reported were looked into, it would be found that they were due to this fact.

The hyperplasia following the removal of the tonsil in adults was not, in Dr. Beck's opinion, an indication that the tonsil had a function; it was not a compensatory hyperplasia. He said that in most cases they did not operate until the third year, but in quite a number of cases the compensatory hyperplasia would follow at or before that age.

He believed, with Dr. Pierce, that otosclerosis had nothing to do with the tonsils as a local process, but he did think that the chronic infection had to do with whatever caused otosclerosis, and he thought that his cases of otosclerosis were better with the tonsils removed than with them present. This opinion was based on twenty-five or thirty cases.

In regard to the anesthesia: in their cases the laryngeal reflexes were never allowed to be abolished, but the pharyngeal reflexes were abolished. Dr. Pierce had probably misunderstood him.

Dr. Beck thought that poliomyelitis in connection with the tonsils should be mentioned in scientific meetings like these, because work had been done by Nuzum and Rosenow, two scientific gentlemen, and we should know about it at least. He had not expressed his personal views on the subject at all; in fact, he had none. He thought that everyone agreed that everybody had his own way of operating, and yet would like to hear about the other fellow's way, was the answer to those gentlemen who think the technic of tonsil operation is a closed chapter. To Dr. Beck it was far from it, and he was changing his technic quite often—however, sticking to the principles very closely.

Dr. Sonnenschein in his remarks had touched upon the point of ligation. Dr. Beck did not see why one did or should not have to ligate a "spurter"; he considered that preferable to going to the patient's room and using more pressure. As to the quickness of operating, he considered that a good point, not on account of the time, but because he thought every operation should be done as quickly as possible. Certain conditions might require half an hour to finish the operation.

Dr. Kenyon had mentioned the aponeurosis. In every case where the Sluder technic is used, one can see the split capsule in which the aponeurosis is separated from the capsule. It matters not what movement is employed.

As to the voice, Dr. Beck had noticed that Dr. Farrell's voice was much better since his tonsils had been removed. Dr. Farrell had said that no case of neuritis had ever been cured by a tonsillectomy. Dr. Beck agreed that no alcoholic or pressure neuritis had been cured, but infectious neuritis was due in most instances to a focal infection from some place, and the neuritis disappeared after the focus was removed; they had had many of those cases, and he was sure that others had likewise.

Paper: Acute Suppurating Mastoiditis Without Tympanitis, Perisinus Abscess. Phlebitis and Streptococcemia—Operation and Recovery.

Dr. Charles H. Long said that acute inflammation of the mastoid process, accompanying or following suppuration of the middle ear, was a common occurrence, but was rather rare when there was an insignificant cause and no discharge from the external auditory meatus. He reported the case of a female patient, aged twelve years, who entered the Postgraduate Hospital on May 5, 1916, suffering from chills, fever, vomiting, great prostration, and severe pain in the left ear. She had had a severe head cold about the middle of April, and later in the month complained of being very chilly. On the afternoon of May 1st, severe pain in the left ear developed, and in spite of treatment the patient grew gradually worse.

On May 5th there was temperature of 104.2° F.; pulse, 120; respirations, 36; no meatal discharge, no tympanitis, membrane red and injected, the nose and throat normal. The ear drum was freely incised, a wick drain inserted, and external dry heat applied to the mastoid. After the paracentesis the pain moved to the opposite side of the head, and continued with great severity until relieved by aspirin. The gauze was removed from the meatus, discolored with only dry blood from the incision. The following two days she was free from head pain but more toxemic, sleepless and delirious. There was great variation of temperature, the maximum being 104.6° F., at noon. She was then seized with pain in the region of the appendix and there was involuntary urination. Blood count gave a leucocytosis of 16,000.

On the 9th she was seen by a neurologist, who diagnosed lateral sinus thrombosis. No X-ray taken. Indications of meningitis were more pronounced.

On the 11th the simple mastoid operation was performed; the inner table was very soft, necrotic, the greater portion being removed with the curette, exposing small pools of pus and masses of exudate which hid the sinus from view. The bone was removed in every direction until healthy dura appeared. With the vein uncovered an examination of the sinus was made with a curved applicator, but it seemed to be free from coagula. To make certain, a long incision was made in the sinus; the blood gushed freely from both ends, no clots appeared, and the hemorrhage was controlled by packing.

On the 13th spinal puncture revealed the spinal fluid normal and not under increased pressure. Following this the patient made an uneventful recovery, leaving the hospital June 3d.

Dr. Long thought that nose and throat cleansing solutions often did more harm than good, and that as prophylactic measures in ear infections they were worse than useless.

DISCUSSION.

DR. GEORGE W. BOOT agreed most heartily with what Dr. Long had said about the use of irrigation of the nose in these nasal infections, and wished to call attention to what had happened at the County Hospital as the result of stopping nasal irrigations in scarlet fever infections. Three years ago it was the rule that every patient with scarlet fever should have the nose irrigated. Two years ago this was stopped, and they had had practically no mastoid operations in the scarlet fever wards since.

DR. L. J. HUGHES, of Elgin, reported the case of a man of thirty-eight years, who was working in a Borden plant in a rather damp place. He had a cold, and complained some of his hearing and ran a temperature for some time. The doctor had suspected a middle ear involvement, but was unable to demonstrate anything positive, and carried the patient along for four or five days, until he developed a tenderness over the antrum. The ear drum was practically normal, had no apparent signs of trouble in the middle ear. As soon as the mastoid showed signs of tenderness he incised the membrane, and immediately pus flowed out under high pressure. Under local irrigation and wick drain the condition cleared up without further trouble, but it was very hard to localize the infection.

It was only when the man developed mastoid tenderness that the doctor was sure of his ground.

Dr. Joseph C. Beck said he had had no experience in such a case as Dr. Long's, but as the doctor was reading his paper he was reminded of a case of socalled primary mastoiditis with sinus thrombosis, where there was no history of a cold or anything in the nose or throat. Chills and fever were present, and there was a suspicion of malaria, because the patient came from the South. The blood examination proved the point that it was not a malaria but a septic process. There was a high leucocyte count, and in that case they waited for a time, because there was not even tenderness about the mastoid, but there was a history that there had been a suppuration in the ear long before. Examination of the ear and hearing were normal. The patient was in the hospital, and they made an X-ray examination which showed a typically sclerosed mastoid. They operated and found a picture much as Dr. Long had described. He thought that in cases where a patient had chills and fever which did not stop, and the blood examination did not show anything to account for it, an exploratory sinus operation should be performed. He thought it would do much less harm to expose the sinus and have a look at it than to wait a week with meningeal symptoms developing.

Dr. Long, in closing, said, in regard to operating sooner than he had, that in the case of a patient whom he saw last week, he made a very strong statement that he would have to operate, and the patient did not return. In the other case he did not have the full history at the time he first saw her; he had no history of a head cold. Dr. Black would see her one day and then would not see her for two or three days—and so it was postponed.

In regard to the malarial cases, there were several cases in the literature where there had been symptoms of mastoiditis, or symptoms simulating mastoiditis, which were due to malaria. Some of them were diagnosed by examination of the blood and no operation performed. Dr. Long knew that some of those cases were operated when the blood was not examined.

ABSTRACT OF THE REPORT OF THE SCIENTIFIC PROCEEDINGS OF THE THIRTY-EIGHTH ANNUAL CONGRESS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

Held at Washington, D. C., May 9, 10, and 11, 1916.

Emil Mayer, M. D., Abstract Editor,

New York.

President's Address.

The President, Dr. G. Hudson-Makuen of Philadelphia, took as his subject:

The Psychology of Diseases of the Respiratory Tract.

While the specialty of laryngology is dependent upon all the various specialties in medicine, its future progress and development depends chiefly upon a knowledge of psychology and its related branch, neurology.

Psychotherapy has been practiced after a fashion since the beginning of the history of medicine, but it has not kept pace with the other forms, the chief reason for this being that the successful practice of psychotherapy requires on the part of the practitioner the profoundest knowledge of both medicine and man, and especially man. Few physicians are mentally and temperamentally capable of practicing psychotherapy, and the improved curricula of the schools do not supply the rising generation of specialists with the assistance which they should have.

Since many of the diseases of the respiratory tract are purely functional and of psychic origin, the laryngologist should be able to distinguish between those of his patients who are psychopaths and neuropaths and those who are suffering from actual organic diseases. He should realize that to treat a purely psychic case by physic measures is not only useless but in many instances absolutely harmful, and to operate merely for the psychic effect of the operation is exceedingly questionable surgery.

Psychotherapy in the form of education and reeducation should always be used, in addition to the necessary medical and surgical measures, for the relief of disturbed respiratory, phonatory and articulatory functions. It is not enough to do operations for the correction of disturbed functions without at the same time or immediately thereafter doing something in an educational way to correct the faulty habits which accompany, either as cause or result, the conditions that we are seeking to modify or cure. An example of the psychophysic habits that arise owing to functional disturbances due to organic lesions is found in the speech of the patient having a cleft palate. When this characteristic speech has developed, no amount of adequacy or efficiency of the palate brought about by operative measures can in itself appreciably change or improve the speech, because the individual accepts his old speech as normal speech.

This principle obtains in all our operations upon the respiratory tract, the object of which is to correct faulty functionation. Its value is apparent in dealing with disorders of speech, the treatment of which differs not from other disorders of the respiratory tract. Formerly, distinguished surgeons operated not alone upon diseases and abnormal structures, but also upon perfectly normal structures, not hesitating to remove cross sections of the tongue and epiglottis, in the hope of curing stammering. To us of the present day this is absurd, for we know that stammering in the great majority of instances is of psychic and not of physic origin, and to cure the affection psychotherapy is quite as important as physicotherapy. What is true of stammering is also true of the other forms of defects of speech.

As the medical profession has been slow in recognizing that stammering and other defects of speech are largely of psychopathic origin and require for their cure psychotherapeutic measures, so have we been slow in recognizing that many forms of asthma, sore throat, and difficult nasal breathing are of similar origin and require similar treatment.

In no specialty of medicine is the importance of these matters so apparent as in our own, for in no specialty is the psychic element so great a factor in the causation not only of functional but of organic disorders as well. In this connection it must not be overlooked that faulty methods of breathing, vocalization, and articulation, although at first of psychic origin, frequently result in organic diseases which cannot be differentiated from diseases having purely physical bases.

A study of the psychobiologic phenomena as they appear in a given individual is merely a study of his reactions to his environment; or, in other words, a study of what has been called his mentation, behavior, and personality.

The new psychology, therefore, teaches us not merely how to treat diseases of special organs, but it teaches us how to treat the patient himself or the reactions of the patient to these particular diseases. A knowledge of this psychology will broaden the scope of our work, and it will tend to make a medical education absolutely necessary to those desiring to practice the various forms of the healing art; but so long as physicians generally disregard this fact, so long shall we have nonmedical practitioners, such as Christian scientists, osteopaths, hydropaths, and all the others of their kind, actually invading our field of operation.

On the Relation of Diseases of the Accessory Sinuses to Diseases of the Eye, Especially in Children, With a Report of Two Cases.

By J. H. Bryan, M. D.,

Washington, D. C.

Diseases of the sinuses occurring in children have been only slightly considered, for the reason that these cavities in the very young are supposed to be so small that there could not be an inflammation sufficiently severe to cause any serious disturbance of the eye.

That these premises are entirely wrong is evidenced by the report of the following cases:

Case 1.—A male, aged eighteen months, had a very marked exophthalmos on the left side following an infection from influenza.

On admission to hospital his temperature was one hundred and four degrees, some secretion flowing from the left nostril, marked bulging of the left eye downward and outward, lids and conjunctivæ were edematous, and the periauricular glands were enlarged.

Seen by the speaker in consultation, the diagnosis of orbital abscess resulting from an infection through the ethmoid cells was made.

The radical operation was then done, the incision commencing at the junction of the middle and outer third of the supraorbital ridge, and was carried inward and downward along the inner border of the nose below its middle. The periosteum along the inner wall and the corresponding parts of the roof of the orbit was stripped from the bone, and in doing so a large quantity of pus was evacuated. The whole of the inner wall of the orbit was removed back as far as the sphenoid. The ethmoid cells were found to be badly diseased, especially the middle and posterior portion, and from the condition found it was apparent the orbital abscess resulted from a direct infection from the middle and posterior ethmoid cells. The amount of pus evacuated was enormous, when we take into consideration the age of the child and the stage of development of these parts at this age. The abscess having been thoroughly evacuated, a strip of iodoform gauze was placed in the orbit back of the eye and brought out through the nose, and a small gauze drain was placed just inside of the inner canthus, and the external wound closed by interrupted sutures.

The child made an uninterrupted and quick recovery, the eye gradually receding soon assumed its normal position. This is the youngest patient the speaker had ever seen with such diseased conditions.

Case 2.—Negro boy, aged eleven years, had bulging of the left eye to a marked degree downward and outward. An absence of pus anywhere within the nose and no signs of caries or necrosis.

X-ray examination showed no abnormality except that the left orbital cavity was apparently filled with a dense mass which seemed confined to the orbit.

Because of all these negative examinations it was believed that there was a growth in the orbit back of the eye.

An exploration of the orbit showed that at the junction of the middle and posterior portion there was a decided bulging of the ethmoid toward the orbit. With a probe the cells were perforated, and a large quantity of pus was evacuated. The whole of the inner wall of the orbit, including all the ethmoid cells, were removed as far back as the sphenoid, and in doing so a large abscess involving the posterior ethmoid cells and the sphenoidal sinus was found. The sphenoidal cavity was unusually developed and filled with thick, creamy pus. All diseased bone and purulent secretion having been thoroughly removed, an iodoform gauze packing was placed in the sphenoid and ethmoid regions, one end being brought out through the nose and the external wound closed by interrupted sutures. At the end of the second day the gauze drain was removed and the nose gently irrigated with a saturated solution of boracic acid.

The patient made a quick recovery, the eye gradually receding within the orbit, and at the end of the ninth day he was discharged from the hospital.

Case 3.—Male, aged thirty-six years, had thrombosis of the cavernous sinuses. Under ether anesthesia he removed the middle turbinals and opened both sphenoidal sinuses, finding some mucopurulent secretion in both cavities. Examination of the secretion taken from the sphenoidal sinuses showed both streptococci and staphylococci present. A lumbar puncture showed the spinal fluid under great pressure, filled with pus cells, roughly estimated at 40,000 per millimeter. Polymorphonuclear leucocytes predominated. The condition of the patient grew rapidly worse and he died.

Postmortem Examination.—The head only examined, showed all the sinuses contained dark and semifluid blood; small hemorrhages on the right half of the cribriform plate of the ethmoid bone. Vessels of the pia mater generally engorged, the basilar surface of the brain showed fibrin and a purulent exudate under the pia, especially on the insula and adjacent opercula. A microscopic examination showed the vessels of the brain engorged. Beneath the membrane was a thin layer of brown material intimately adherent to the brain substance beneath. On section the brain substance showed brownish points near the surface, the brown exudate under the membrane showed fibrin, red blood cells and mono- and polymorphonuclear round cells.

DISCUSSION.

Dr. Lewis A. Coffin, New York City: Dr. Bryan's experience is unique, I think, in the extreme youth of his first patient—eighteen months old. My youngest patient was six years old.

I have reported a case before this society in Boston, a case of thrombosis sinus cavernosus, upon which I had operated. That case has been very instructive to me. I think we lose sight of the fact that these ethmoidal veins for the most part empty into the ophthalmic veins. Now, if we know anything at all about a thrombus and its cause, where is there a more favored place for it to develop?

Then there is another thing—when we operate those cases, they bleed very profusely. Why other cases do not bleed the way they do, I cannot tell. I am reminded of the case of a girl who was absolutely blind in one eye four weeks, with a choked disc. I did a radical operation on her, and it was absolutely impossible for the man with me to sponge it so that I could see from the field of operation. I have had several of those cases of serious bleeding, just the same as if you put a stricture around the leg.

To return to that case of cavernous sinus which was so interesting and instructive to me. Dr. Coakley saw that patient in consultation with me, and we both felt acute sphenoidal sinuitis was present. Another consultant said: "We think you have probably saved her life by the great bleeding."

What I am going to do now with these cases is to give treatment in the shape of great big doses of lemons and lemonade.

I recall one case which was referred to me in which I really could not make out much sinus trouble, and I told the eye man to put her on big doses of lemonade and keep her on it, and the eyes cleared up. Of course, the whole thing is to reduce the coagulability of the blood. Finally, we must work out the relationships of this circulation.

Dr. Cornelius G. Coakley, New York City: I saw a child two years of age, a robust child, with a swelling around the left orbit, exophthalmos, protrusion of the conjunctiva and swelling down on the face, with a serosanguinous discharge from the left nostril. Bacteriologic examination of nasal discharge showed streptococci; the variety, however, was not worked out. Child had an acute nephritis with a marked amount of albumin and casts—hyalin, granular and epithelial. There was also an endocarditis of sudden onset; the abdomen was also swollen. It looked very much as though it was only a question of time before the child would die, and we all thought it best to try and open up the cheek. Following that the stools had a large quantity of bloody, purulent discharge, and of course the prognosis was extremely bad, for it looked like a general infection. A blood culture was taken and the following morning was reported upon as being very markedly positive. A blood transfusion was done from the father to the child. I expected to hear of the death of the child within three or four days, and two weeks later I heard the child was perfectly well. I think the point is most valuable in the treatment of these cases. On account of the general condition, I advised against sewing up of the orbit.

Some time previous to that, I saw a child of five years in consultation. This condition followed one of the exanthemata. That child was later operated upon.

I also saw a case last winter of cavernous sinus thrombosis, one most interesting as to the source of infection. The patient was a man who had not been feeling particularly well, and a diagnosis of carious teeth and abscess of a tooth was made. He went to the dentist with that diagnosis. This was not the diagnosis of his own dentist, but had been made by another dentist, and the diagnosis was disputed. Fortunately, the dentist who made the diagnosis had taken radiographs of the teeth and sinuses, and I was much surprised at the good radiograph he obtained of the sinuses. This was a week before I saw him. The frontal sinuses, ethmoid, sphenoid and antrum appeared perfectly normal. He showed me what he called an abscess, which may have developed a swelling around the orbit and along the eye on the opposite side from where a molar tooth of the left upper jaw had been removed. The swelling first began in the right eye. We put a hole in the antrum and drained it and a few days afterwards the opening was practically dry.

I saw the patient of whom Dr. Coffin spoke. I think we have two types of cavernous sinus thrombosis—the type that is infected, and the type that is inflammatory. If you get an infected cavernous sinus thrombosis, you are bound to have it end fatally; but if you get one in which the organism is very mild, why there is a possibility of recovery.

Dr. Hanau W. Loeb, St. Louis: I would like to make a suggestion with regard to the statement made by Dr. Coffin as to our lack of knowledge of the venous relationship. We might of course learn the names of each trunk, but I think we would find that we would not be any better off. But we do know there is a very abundant venous distribution in this region, and having that abundant venous distribution, we naturally have results of infection in the neighborhood, and that undoubtedly accounts for the majority of infective conditions in the orbit coming from the nose. However, there is such a thing as a condition resulting from mere nearness to an acute inflammatory condition. For instance, we know that acute edema results in the larynx from inflammation in the neighborhood. A year ago I reported a case before this society in which blindness had resulted from an acute ethmoiditis, and the blindness disappeared within a week after operation upon the ethmoid. At that time, too, I called attention to an observation made from studies in the neighborhood of this region, that when the posterior ethmoidal cells replace the sphenoid, the optic nerve, instead of being at a considerable distance from the ethmoid, runs along the lateral wall of the posterior ethmoid cells. This so happened in three out of thirty cases I had examined. Now, there is a further observation which I might make, that when we do have a condition of acute inflammation of the optic nerve, it is because the posterior ethmoidal cells have replaced the sphenoid, and the nerve, instead of running at some distance from the sinus, runs right along from the lateral wall.

Dr. Bryan (closing the discussion) had nothing to add.

Sore Throat Clinically Considered.

By Samuel Johnston, M. D.,

Baltimore.

In the clinical study of "sore throat" we should scan the physiognomy of the patient, mark well any changes in the voice tones, and note the odor of the breath before entering into a more detailed examination of the case.

Among the conditions causing changes of the voice may be mentioned paralysis of the soft palate, defections in the conformation of the palatine arch, swollen tonsils, benign and malignant growth in the nasopharynx, laryngeal inflammations, paralyses, and so forth.

The odor of the breath may call attention to such conditions as uremic poisoning, pulmonary gangrene, ozena, necrosis of the nasal bones, and so forth. The need of careful inspection of the lips, gums, teeth, tongue, palate, pharynx, nasopharynx, lingual tonsils, epiglottis and larynx is emphasized.

In examining the nasopharynx an ulcer, usually of an infectious nature, is found here when least suspected, and in infectious diseases sore throat is by no means uncommon.

The writer's experience has proven that diseases of these regions differ in no way from similar pathologic changes in other parts of the body, and should receive the same therapeutic and surgical treatment.

Conservative and mild measures, however, should be the rule and guidance.

DISCUSSION.

DR. LEWIS A. COFFIN, New York City: As we grow older I think we all get to such a position where we feel that perhaps as younger men we interfered too much with the architecture of the upper air passages.

The Diagnosis and Management of Vasomotor Disturbances of the Upper Air Passages.

By J. L. GOODALE, M. D.,

BOSTON.

In a large proportion of vasomotor diseases of the upper air passages the disturbances are dependent upon the entrance of a foreign proteid into the system. The method of entrance may be through contact of the proteid in question with the mucous membranes of the respiratory or of the gastrointestinal tract, by inhalation or ingestion, respectively. Foreign proteids may perhaps also develop in or upon these mucous membranes through autolysis of pathogenic or saphrophytic bacteria. The application of the skin test to these conditions is of diagnostic value when employed with a recognition of the phylogenetic relationships of animals and plants as determined by studies in serobiology.

Proteid material for testing should be prepared both from the keratin and sera of domestic animals, from the pollen of the chief causes of hay fever, and from the various articles of food which enter commonly into the diet. Bacterial proteids derived from the various invaders of the respiratory tract should be available, either in solution or in the soluble form.

When the skin reactions to the various classes of proteids have been determined, the management of cases will depend largely upon the relative preponderance of the local reactions in relation to the clinical history. If the cause is found to be seasonal, as in hay fever, immunizing treatment by injection of pollen extracts is likely to prove of service. The sensitization returns during the following winter, and treatment must probably be repeated annually. If the cause is perennial and is due to inhalation of foreign proteids, it is wiser to avoid the cause rather than to seek to effect a cure by immunization. If the disturbing proteid enters into the ordinary articles of diet, a tolerance may be gradually established by feeding the substance in progressively increasing doses. Disturbances of bacterial origin have not yet been sufficiently studied to enable the formulation of a definite plan of treatment, but the results of these investigations confirm our present methods of treatment, and emphasize the importance of draining regions which can retain the products of bacterial activity. Septic foci should be removed. Vaccin therapy is likely in such anaphylactic cases to be more accurately guided than in the ordinary individual.

DISCUSSION.

DR. JOHN F. BARNHILL, Indianapolis: I would just like to ask one question with regard to whether the matter has been brought down to a working basis. For instance, if a patient comes to Dr. Goodale, what plan does he adopt for determining what the sensitization may be? Has he come to any conclusions?

DR. ROBERT LEVY, Denver: We were very much stimulated by Dr. Goodale's work last year. I should like to ask him two questions. First, what is his mode of procedure when we have a multiplicity of sensitizations? I would like to have that more at length. And secondly, having determined for a certain individual his sensitizations to a number of proteids or inhalations (I have particular reference to inhalations), what method does he proceed with in the treatment of these individuals?

DR. HANAU W. LOEB, St. Louis: I would like to ask Dr. Goodale to tell us if lactate of calcium and calcium chlorid salts have, in his experience, had the effect of reducing the sensitization.

DR. J. L. GOODALE, Boston (closing the discussion): If I had read the whole of my paper, all of these questions would have been answered. Answering the first, as to how we proceed, I think the best plan is this: See whether the symptoms are present throughout the year, or whether they come at a definite time. If seasonal, they are probably due to something in the air; most commonly, of course, pollen. If they may occur at any time during the year, we inquire as to whether they stand in relation to colds or conditions of acute infection of the bronchial tubes. If you see the individual is entirely well throughout the summer and winter, and once in the fall or spring has a severe cold and develops asthma, we can, under those circumstances, rule out cases of foods that he takes into the stomach, and also rule out the question of animals; consequently, it is one of these other products. On the other hand, it may not be fully a seasonal infection in that it may come in November or January, or at whatever time it may be, and in that case I should proceed to investigate with special reference to bacterial proteids, and look for those organisms that we know may invade the mucous membranes of the upper air tract. Now, if the symptoms are throughout the year, without much influence from season or travel, or various modifying circumstances, I should look for asthmatic animals—especially in the case of children—cats and dogs. Children don't know cats or dogs trouble them, but they may have asthma entirely due to that. I have a recent letter from a man whose daughter had been the victim of asthma when four or five years old, who looked like a little old woman. It was really pathetic. I found it was due to nothing but the cat, and we kept the cat away. The little child has now resumed the bloom of youth and the parents are very greatly gratified.

That is the manner in which you proceed. You bring the season, the occurrence, the incidence and mode of development of the symptoms into relation with the patients, and that simplifies it very much indeed. The use of lactate of calcium and the chlorid has made no material difference.

Angioma of the Larynx.

By Emil Mayer, M. D.,

New York City.

This affection is of very rare occurrence, the writer having found only forty recorded cases in the world's literature.

He presented the history of a woman, aged fifty-two years, who had a history of previous attacks of laryngitis with hemoptysis, and who had a tumor in the larynx on the left side, extending from the left false cord and covering the true cord on that side.

The diagnosis of cancer of the larynx had been made by laryngologists who had seen her previously.

The patient was admitted to Mount Sinai Hospital, and the writer asked to report upon her condition, and remove a portion of the growth for diagnosis, if necessary.

Owing to the yielding character of the growth, its bluish tinge, and the history of previous bleeding, the diagnosis was made of angioma of the larynx by Dr. Mayer. Removal of a portion for diagnosis was deemed too dangerous, and external operation was advised. This was subsequently performed by Dr. C. A. Elsberg.

The growth was removed, the mucous membrane sutured, and the pathologist reported it to be a hemangioma.

The patient made an uneventful recovery.

The writer concludes that angioma of the larynx is a rare disease, occurring mostly in adults, the proportion of males to females being about four to one. It may be mistaken for cancer. Endolaryngeal removal, even of a portion of the growth, for diagnosis is fraught with danger, while laryngofissure is entirely safe and feasible.

DISCUSSION.

Dr. Henry L. Swain, New Haven: If Dr. Mayer wants another case simply to bear out what he said about the danger in these cases, I might mention one in which I attempted to remove a growth of this kind. We got hemorrhage all right. If anything further was needed to substantiate the diagnosis, the microscope did. I have a slide at home showing it to be true angioma of that type.

I am interested to hear what Dr. Lynch thinks he could do with such a case by suspension.

Dr. Robert Clyde Lynch, New Orleans: I have never had an opportunity of seeing one of these cases, and have, therefore, had no personal experience with them. I think, however, that if it were possible to get underneath the place, and if the surface was not too broad and the position properly localized, it might be possible to do it under suspension. However, that could be decided at the time the case presented itself.

After seeing the illustration of the size and site of this angioma, I would not attempt its removal under suspension.

Dr. Emil Mayer, New York City (closing the discussion): I merely wish to pass around this illustration showing the shape and site of the angioma.

Further Progress in the Use of Radium in the Field of Laryngology.

By D. Bryson Delavan, M. D.,

New York City.

Decided advances have been made during the past year in our knowledge of the application of radium in diseases of the upper air passages, while the number of the conditions in which it is found effective is being steadily increased. Several institutions, notably the General Memorial Hospital of New York, have been fortunate in acquiring amounts of radium large enough to meet all probable demands; while those administering it are gaining experience in its use and learning how it may best be utilized and controlled.

Uses of Radium.—Closely allied to our department may be mentioned the success of radium in "vernal catarrh," or that form of conjunctivitis which occurs during the spring, and often, when established, lasts throughout the year. Abbe has observed ten cases, recurrent for many years, and asserts that improvement always begins soon after the first treatment. Even patients who had been previously treated by the removal of hypertrophied masses, cauterization, and caustics received uniform benefit from radium and were ultimately cured.

A second interesting condition in which radium has no rival is in the reduction of lymphoid tumor cases, as found in tumors of the tongue, called hemolymphangioma.

The treatment by radium of papillomata of the larynx, as with warty growths in general in other parts of the body, is being attended with ever increasing success.

Leucoplakia of the tongue, Abbe believes, is as capable of cure by radium within the mouth as is the skin hyperkeratosis. In the mouth, however, the duration and the method of application require more judgment and skill to attain good results. The treatment is associated with transient painful irritation, but this seems essential to success. Chronic abrasions and fissures of the lip are curable by radium.

In the treatment of nevus, excellent results are being obtained.

New growths of nonmalignant type are receiving an increasing amount of attention, with excellent results. Thus, Abbe has shown a case of myeloid tumor of the lower jaw completely cured.

Tumors of the larynx of various kinds have been caused to disappear, with complete return of the singing voice.

In the field of nasopharyngeal fibroma the use of radium is most encouraging, particularly so in view of its success in the treatment of fibromata of the uterus.

Two cases observed by the writer, of sphenoid carcinoma, are worthy of notice. Both originated in the left side of the throat, close to the wall of the larynx, probably extralaryngeal. Both were seen late, long after operation would have been possible. Both patients were men in the early fifties, hitherto in perfect health, active, vigorous, and of good antecedents. When first seen, the disease in both had invaded the interior of the larynx, the left lateral wall of the pharynx, the pyriform sinus, the tonsil, and the base of the tongue. In both, ulcerations were present and there was marked aphonia and dysphagia. Both were treated at the same institution, exposed to large doses of radium, and in both the results were materially the same. The first effect locally was an almost immediate control of the secretions of the throat. From having been abundant and fetid, they promptly ceased. Following this, the areas of ulceration rapidly diminished in extent, and in the less severe of the two cases disappeared, while in the other case they seemed to do so. The swellings, which had appeared over extensive areas of the affected parts, decreased markedly, and the infiltrated tissues were reduced in size, became soft to the touch, and more natural in appearance. Meanwhile the voice became clearer and deglutition improved so that both patients were able to swallow without pain and to largely increase the variety of their food. The general improvement was remarkable. Digestion became normal, and sleep more prolonged and restful. Strength increased steadily, and there was an almost normal condition of good spirits. One patient, a physician, was able to resume office practice and operative work for a period of over two months. Both have agreed that if the further progress of the disease should be entirely unfavorable, the benefit gained in the relief of suffering and the added comfort afforded would well have repaid them for any inconvenience which the radium had caused, whether from burns of the skin or from any other result.

Already results worthy of profound consideration have been obtained. Far from being discouraged, there is every reason why persistent and continued effort should be made to finally solve the existing problems and give to the world a successful cure.

DISCUSSION.

DR. CORNELIUS G. COAKLEY, New York City: The great trouble with the use of radium is that one can find little or nothing to be used as a guide in the dosage of this material. Dr. Delavan mentions in his paper "the application of a tube of strong radium for fiften minutes." Now, that does not mean anything to those working in it. I would like to know the actual amount of that to be a guide to other men.

Another great difficulty with the use of radium is the enormous expense of the material. At the present time we are having placed at our disposal by the Crocker Institute about one hundred grammes of radium, the cost of which is somewhere over $10,000. We use it on various pathologic conditions. That is a prohibitive cost for most men in the practice of medicine, so they never use it.

Our greatest difficulty is that it burns, and it burns very severely. We have not yet been able to properly screen this material when placed in the mouth, or in the nose, or nasopharynx, or on the exterior of the body so as to have it reach the object of the pathologic process without getting more or less burning. If you use too little, you do not get enough action; if too much, you get terrific burns which occur not at the time; but even months later, as happened in one case treated by radium at Johns Hopkins. The patient had an application of radium a year ago last March, and the appearance of the burn did not manifest itself until August of last year. In one of my own cases in which we applied it, a portion of the nose had been burned off as a result of the action, the burn coming on six months after the application. The radium destroyed the growth of the nose, but also almost all of the nose as well. It caused external thrombus and finally destruction.

Those are some of the things with which we have to contend. We know nothing ourselves, and there is very little in the literature to guide us in escaping those factors.

There is one other point in relation to malignancy. If you use it you will unquestionably stimulate the action of the malignant process in the same way as you would if you curette or cauterize it. If you are going to use radium, you have got to get it right into the mass and have an amount which the ordinary practitioner cannot afford to invest in, so that you get enough of it to do more than merely stimulate the growth.

Dr. Jacob H. Hartman, Baltimore: There is a case I would like to report in this connection, a most unusual experience.

In August, 1913, there was a patient, a gentleman, sent to me by his family physician with an intralaryngeal growth, of a red, angry color, filling up a large part of the larynx and interfering with the respiration. I gave him an unfavorable prognosis and advised him to have another opinion. He was taken to Dr. Winslow for consultation, who agreed that the growth was undoubtedly malignant in character, an inoperable case. On September 4th two small portions were removed very carefully for pathologic examination. The microscopic examination was made and the specimen reported malignant. On September 14th a tracheotomy was performed. On September 21st radium was applied externally for three hours; I do not know which rays, though I believe the gamma rays —at any rate, the most penetrating. On September 24th radium was again applied for nine hours, and was followed by most extensive burns all over the neck, and the patient suffered intensely. On October 6th there was almost complete obliteration of the growth, and on October 23rd the patient was again examined and there was no vestige of the growth at all, only a slight thickening of the mucous membrane over the arytenoid space. The larynx is absolutely normal. The last time I saw him, April 12, 1916, the larynx was thoroughly clean, and there was no evidence of growth noted at all. The patient had gained thirty to forty pounds and was remarkably well and absolutely free from any trouble of this kind.

Dr. Harmon Smith, New York City: About three years ago a young boy, twelve years of age, who had a large growth, was sent to the General Memorial Hospital in New York City, where we now have some of the strongest radium in this country. At that time the application of radium only increased the growth, but we had not the strength of radium as we have at present. The boy was sent to me and had the growth removed on two different occasions with considerable hemorrhage, nearly fatal, each time. Injections of ammonium chlorid and trichloracetic acid failed to hold it in check. The condition progressed gradually, the boy became anemic, and the growth extended out into the jaw and filled up the cheek. Some weeks ago he was sent back to the General Memorial Hospital, where they applied radium once for a period of three hours. Following that he had a hemorrhage, and a few days after the hemorrhage an edema of the larynx set up which necessitated tracheotomy, and the boy died as a consequence of it.

I recall a case of similar character where removal had not completed the disappearance of the growth and it had recurred, so this young boy, about nineteen years of age, was sent to have his application of radium at the same time. He had an application of strong radium which set up such inflammation in the cheek and the tissues adjacent to the growth, that for a space of at least a week the boy was unable to swallow at all. We had to feed him by rectum and it became a very serious proposition. So that, in my opinion, we should know more about the action of radium before applying it indiscriminately. Yet this radium was in the hands of an expert, and applied at a hospital where radium is being used experimentally as well as practically.

I also had three cases of cancer of the larynx which required tracheotomy, and a tube of radium was inserted to be left as long as was seen fit. Those cases went on without any apparent benefit to death.

Dr. John R. Winslow, Baltimore: It was my privilege to see the case mentioned by Dr. Hartman from the beginning until the end. The last time I examined him there was no evidence of the growth, nor any indication of the site of attachment of the growth. One who had not seen the case previously would have been unable to determine whether there had ever been any pathologic condition in the larynx. Of course, the case is an absolute cure of a condition which, as far as my experience and judgment go, would have been an absolutely incurable and inoperable one.

An interesting thought occurs to me as to what happens to these growths. I took particular pains to inquire of this patient whether he had had any increased expectoration or secretion in any way pointing to a breaking down by ulceration, or similar process, and he admitted no such condition. The growth simply apparently disappears.

Dr. Cornelius G. Coakley, New York City: I would like to ask, with reference to Dr. Hartman's case, whether the growth of the larynx was ulcerated, and what was the strength of radium used.

Dr. John R. Winslow, Baltimore: We are, however, greatly handicapped, for while we have practically one of the largest collections of radium in the country, it belongs to an incorporated, private institution which is conducted largely on a commercial basis. The cost of an application of radium is somewhere in the neighborhood of $100, if not more. That, in itself, is almost prohibitive in many cases, so that it can only be used exceptionally. There is no one who has obtained a loan of radium, and so far as I know there is no government institution for this purpose. It seems to me it is a field in which the government should take a hand and incorporate a national radium institute for the benefit of the public.

Just before I came away I saw a case of epithelioma of the nose, completely filling the nose and invading the ethmoid, as well as causing protuberance of the eyeball, in which I expect, if possible, to have the radium applied. It is a case which is absolutely inoperable. The patient is seventy-nine years of age, and the tumor myxomatous in appearance, bleeding on touch. While there has been no opportunity of removing a specimen for examination, I have no doubt but that it is an epitheliomatous growth.

Answering Dr. Coakley's question as to whether the growth in the larynx was ulcerated, and the strength of the radium used in Dr. Hartman's case, there was no ulceration and no involvement of the glands. I do not know in what strength the radium was used, but I know it was very strong.

Dr. Robert Clyde Lynch, New Orleans: I have had two or three experiences with the use of radium. The first case was that of a patient with sarcoma of the nose, treated two years ago. I did the first stage of the Caldwell-Luc operation and put in the radium, unscreened—simply the little capsule with the glass container which holds the radium into the nose and antral cavity. This was a fifty-five millimeter tube. The diagnosis of sarcoma had been made by microscopic examination, and the nose had been blocked up completely. In the course of four weeks there was free nasal respiration, and an examination with the Holmes laryngoscope did not reveal any evidence of the growth at all.

The second case was one of an intrinsic laryngeal growth in which an extension occurred, necessitating something else being done. Seventy-five milligrams of radium were applied externally, fifty on the diseased side and twenty-five on the opposite side, the radium being screened with brass, then four rows of sheet lead, then the same thickness of aluminum, then gauze, and finally a rubber cot. This roll was applied to the outside of the larynx and left for twelve hours. Two days following the application there appeared the most beautiful sunburn, the larynx was extremely red and the patient complained a little. The growth became pale, white and dry; it gave the appearance through the mirror of being dry. There was a rest of five days and then a reapplication for eight hours, accompanied by a recurrence of this burn, though not quite so much. Then there was a rest of one week and a reapplication for twelve hours. After this there was an apparent retraction of the growth. The surface remained dry and gave one the impression as if the deeper tissues of the growth, that is, the deeper invasion of the growth towards the cartilage, was becoming less. I have devised an intubation tube for the intralaryngeal application of radium. In reporting an application of radium, I think we ought to lay special stress upon the size of the dose and the length of application.

DR. HENRY L. SWAIN, New Haven: In a case exactly similar to the one cited by Dr. Winslow, in which he is expecting to use radium, we used the capsule as mentioned by Dr. Lynch. In this case we merely put the capsule into the substance of the growth, and it bled profusely for a little while. The capsule was allowed to remain in over night and the patient sent home in the morning. An external burn resulted from the application and marked recession of the growth. Dr. Winslow said he was interested to know what became of these growths. This was one of those pale, flat growths, and it became rosy hued, and all of this rose hued area eventually disappeared by mild ulceration. The whole growth was not exposed to this application of radium, so a second application was given about six weeks after the original application, at this time the radium remaining for nearly five hours. It is now only three months since the second application and the growth has practically disappeared; at least we cannot find any trace of it with a careful search. I think it is extremely valuable for us to recite our experiences in this way and discuss them from so many angles.

DR. CORNELIUS G. COAKLEY, New York City: Seventy-five milligrams of radium, when put into the growth and left there, will cause an enormous amount of destruction. Seventy-five milligrams applied on the outside of the neck for involvement of the larynx, or tonsil, or pharyngeal mucous membrane, will not do very much. You must have an enormous amount, four hundred or one thousand milligrams, if you wish to get results from external applications through healthy tissue to malignant tissue. This must be left in about three or four hours. I think the best results are obtained by strong doses applied for a short time, and then reapplied at short intervals of about two or three days.

DR. D. BRYSON DELAVAN, New York City (closing the discussion): There are some things which belong to the laboratory. Radium does, because it requires large amounts to produce effects. The general practitioner has no more to do with radium than the driver of a hackney coach with the New York Central Railroad. It is something the general practitioner cannot, in the nature of things, know about. It is something that even Dr. Abbe, who for many years has spent such a large amount of study upon the subject, does not know enough about yet to specify. It is something which is so new and the discussion of which is so fresh that of course there is no literature to guide us. There is nothing which we can use as a guide but the work being done in a few places by a few men.

The largest amount of radium in this country is at the laboratory with which I have the honor of being slightly connected. There are two other institutions in New York doing the best work they know how to do and trying hard to learn just how to do the work—the one to which Dr. Coakley referred, and the other is one under Dr. Bissell at Johns Hopkins. Our institution will soon have two grammes of radium, and I don't know any place in Europe that has as good a supply. Radium has not yet proved a cure for cancer, but it has a possibility, and that possibility is worthy of following. Radium will burn. Well, it hurts to be operated upon in any way. It is not pleasant to take ether and be laid up for a month or two, or five months, after painful surgery, but it is a very beautiful thing to be well and among your friends. And so it is with the use of radium. Suppose it does burn? In those two cases quoted, both patients suffered severe burns, but they rejoiced at it, even though they did not result in cures, but only in temporary benefit.

If there is nothing in it, why we have tried and done our best; if there is, there is a great prize in store.

An Epidemic of a Severe Form of Acute Infection of the Throat, With Abscess Formation. Report of Fifty-eight Operations.

By Clement F. Theisen, M. D.,

Albany.

This epidemic occurred in Albany during December, 1915, and January and February, 1916. Cases occurred in all parts of Albany, three hundred and eighty-four coming under the writer's personal observation.

Fifty-eight of the patients developed abscesses in different parts of the fauces. Forty-four of this number were more or less typical cases of peritonsillar abscess. Of the remaining fourteen cases, in eight, abscesses developed in the lateral columns of the pharynx. There were two cases of infection of the epiglottis with great edema and some pus, two cases of abscess of the lingual tonsil,and two of retropharyngeal abscess. Joint complications, acute arthritis and polyarthritis occurred in twelve cases, acute endocarditis in one, and in twenty-four, examination of the urine showed the presence of albumin and casts. In sixty-eight cases acute otitis media, requiring incision of the tympanic membrane, developed, with one mastoid complication in a child in which the membrana tympani ruptured ten days before the writer was called.

Cultures taken during the epidemic showed streptococcus infections in the majority of the cases. A few were pneumococcus infections.

The milk supply was probably not a factor in this epidemic.

There were no deaths among the writer's cases, although some patients were seriously ill, particularly those with joint and kidney complications.

The onset of the attack in most cases was extremely severe, with chills, great prostration, swelling of the glands of the neck and high temperatures, particularly in children.

Many cases that did not go on to abscess formation, could not be differentiated clinically from follicular tonsillitis. In some there was the distinct ulcerative type of acute tonsillar infection, and these cases were very ill. In a small number of cases a distinct exudate covered the tonsil, but none of these cases proved to be diphtheria. Great edema and intense acute angina were characteristic of many of the cases. The laryngeal mucosa was involved in the edematous process in a small number.

DISCUSSION.

DR. HENRY L. SWAIN, New Haven: I feel that I must say that Dr. Theisen did not lose a single case of his four hundred cases.

DR. CLEMENT F. THEISEN, Albany (closing the discussion): I would like to mention that the epidemic in Albany and the adjacent city was not due to milk. We studied all the epidemics with that point in mind, and proved that fact conclusively. There have been epidemics of sore throat caused by infected milk, but in the epidemic in Albany this could be ruled out, for the cases were distributed in all parts of the city, and there were just as many supplied by one dairy as by another.

A Résumé of My Year's Work With Suspension Laryngoscopy.

BY ROBERT CLYDE LYNCH, M. D.,

NEW ORLEANS.

To prevent fracture of the alveolus the writer places a strap under the occiput and clamps it into the angles of the pear-shaped ring, thus relieving the pressure of the tooth plates against the teeth.

In order to prevent other tooth injuries, the writer uses dental impression spoons filled with moulding compound which offers complete protection to the teeth, facilitates the introduction of the spatula, and makes it easier to keep the spatula in the middle line.

He also described the table which can be raised twenty inches, the top can be tilted and can be moved in a circle. There are foot and shoulder braces.

Regarding intrinsic epithelioma of the larynx, the writer's experience has been exceptionally good with endolaryngeal removal, but feels that his experience is not large enough to reach definite conclusions.

Dissection under suspension is not difficult, and can be done without permitting an instrument to touch the tumor mass.

Nineteen cases of papilloma have been successfully operated, dissecting well below the base, curetting and painting with alcohol.

Pedunculated fibroma, vocal nodules, a cyst of the aryteno-epiglottic fold, pachydermic laryngitis, perichondritis of the thyroid, fracture of the thyroid cartilage, tubercular laryngitis, abscess of the epiglottis, and foreign bodies in the trachea and esophagus were all cared for by means of the suspension apparatus.

DISCUSSION.

DR. EMIL MAYER, New York City: There is one phase of suspension that has not been mentioned by the writer of the paper, which has worked splendidly in my hands, and that is in the treatment of stenosis of the larynx and trachea. In one instance, that of a little boy who was on my service at the Mt. Sinai Hospital, and who had tracheal stenosis, postoperative, it became necessary to intubate, and the only way possible was under general anesthesia. It was my hope that we could keep the trachea widely open with the wearing of the intubation tube. With the capable assistance of my associate, Dr. Yankauer, this boy has been anesthetized, and we intubated a number of times. He has been wearing the intubation tube for a matter of two years now, and we are in hopes that he will be able to do away with it eventually by the interesting method of transplanting fascia in cicatricial tissue. In this case I do not know what I would have done without being able to put the child under general anesthesia and use the suspension apparatus; first of all, to distend the web of cicatricial tissue, and then to intubate.

The second case was that of a man with syphilitic stenosis of the larynx. It became necessary to do a tracheotomy on account of the stenosis. He then received regularly, about three times a week, a distension of his larynx by means of Schrolter's tubes of increasing diameters, so that by the time he was ready for the intubation tube, his larynx was fairly well distended. He also received some salvarsan injections. I felt that we were able to do something for him in a good deal quicker time than ever before in my experience. The man was given a hypodermic of morphin and told what we wanted to do, and without any general anesthesia whatever we put in the intubation tube. This remained in situ, and about ten days afterwards he coughed out the tube. The amount of breathing room was so much greater directly after this, that we were willing to do without the intubation tube. In this exceedingly short space of time, by this means and the treatment of salvarsan and mercurial injections, he has been quite cured. The suspension apparatus helped us materially in this case.

DR. JOHN F. BARNHILL, Indianapolis: I would be glad if Dr. Lynch would tell us about his present views of anesthesia with the apparatus.

DR. ROBERT LEVY, Denver: In the treatment of tuberculosis of the larynx I have been particularly pleased with its value in removal of the epiglottis; the special feature in its favor here is that one has perfect control of the hemorrhage following the excision of the epiglottis. Before the advent of the suspension apparatus, in an attempt to remove the epiglottis, I had rather serious hemorrhage upon two occasions. In several cases of epiglottectomy since the suspension work has been in vogue, hemorrhage has had no terrors whatever for me. I have seen an artery spurt from both sides of the stump, of sufficient size that it could be seen by a class of students at some distance from the operation. The artery spurts into the mouth, and one controls the hemorrhage with the greatest ease by means of artery forceps.

In curettage or cauterization of the larynx for tuberculosis the apparatus is of great value because you can do so much more in one sitting than by the direct method.

With these cases it has been my practice to use scopolamin and morphin, although in one case I had a death on the table, which I attributed to the highly unstandardized preparation of scopolamin.

Tumors of the larynx have been exceptionally easy of removal with this method. This fairly sized malignant tumor was removed by suspension. It sprang from the posterior wall of the larynx and came over by its attachment on the external surface, so that it was partially extrinsic. It was placed in such a position that when the patient sat erect it flopped into the larynx and produced dyspnea. On two occasions the patient was picked up on the street and sent to the hospital, so that any prolonged operation by direct method would have been impossible, but by suspension it was removed with the greatest ease.

In the dissection of a venous cyst with large veins that bled freely in the hypopharynx the method was particularly valuable, because I could make a very wide dissection. This case was one in which removal had been attempted on several occasions without success. I made two attempts, a cautery had been used several times, and one consultant injected it with iodin. It recurred always until we were able to make a wide opening under suspension.

DR. HARRIS P. MOSHER, Boston: When Dr. Lynch began his work, especially the work of removing malignant disease of the larynx, I withheld my judgment for a time, waiting to see what his results would be. In the paper read this morning I was very pleased to see that he had gone on carefully with a great deal of judgment, and that he had come to the conclusion which many of us felt that he would come to—that is, that the removal of malignant disease by suspension was not a final success, but that thyrotomy was better. For myself, I feel that I much prefer thyrotomy to suspension removal of malignant disease.

DR. ROBERT CLYDE LYNCH, New Orleans (closing the discussion): The chief essential of the suspension apparatus is to obtain relaxation of the parts. Whether the anesthetic be morphin and scopolamin or another, is left to the individual judgment of the case. I have used only cocain anesthesia, and prefer it. I give one or two doses or morphin, one-fourth grain one or one and a half hours before the operation, and one-eighth grain one-half hour before the operation is to be done. For surgical work I much prefer general anesthesia in those cases where it is not definitely contraindicated. Under general anesthesia we get the proper amount of relaxation of the parts, and are able then to suspend the patient until we get the proper view. You must understand, suspension is not an easy proposition so far as the patient is concerned. It is not painless, and for that reason patients must be put in such shape that will will not interfere with the work after it has been once started.

Pathogenesis of the Nasal Neuroses.

By Charles P. Grayson, M. D.,

Philadelphia.

The subject is reintroduced because of the fact that during the past few years some very important additions have been made to our knowledge of the causes and consequences of certain disturbances of metabolism, the effects of which should arouse particular interest because of their bearing upon the genesis of the nasal neuroses, being those that are exerted upon the cerebrospinal and sympathetic nervous systems. Reference is intended only to the vaso- and secretomotor neuroses of the nose, and not to the functional disorders of the olfactory nerve, nor the neuralgias of intranasal or accessory sinus origin. The treatment of these affections has not been rational, in the sense of having been directed against their cause rather than their effects. It has been too largely local. Loss of vasomotor equilibrium is almost invariably due to a greater or less degree of nervous exhaustion. Anaphylactic reaction may explain a moderate number of cases. The basic condition of almost all of these patients is one of neurasthenia due to metabolic derangement occasioned by persistent violation of the laws of personal hygiene. The studies of Abderhalden, Fanser, Kafka, and others, of the physics and chemistry of the colloids and of the effects of ferments, toxins and hormones upon the sympathetic nervous system have a close relation to the etiology of the nasal neuroses. No strict pathogenic analogy exists between these diseases and the angioneuroses of the nose, but it requires no great stretch of the imagination to discern a possible or even probable relationship between the metabolic processes concerned in each. It is almost a certainty that in dealing with the nasal neuroses we are dealing with phenomena that are the outcome of some perversion of metabolism. Its exact nature may vary in different cases, involving at times some glandular disfunction, at others toxemias of intestinal origin, and at still others the presence in the blood plasma of certain ferments or hormones, but in all cases the symptomatic result is due to the same intermediate cause—a toxic action which expresses itself through the sympathetic or autonomic nervous system. To be successful in suppressing the neurotic type of nasal disorder, we must abandon our fruitless search for specific remedies and apply ourselves to the hygienic instruction of our patients and to the development of an internal treatment founded upon a recognition of the essential nature of the disease.

DISCUSSION.

Dr. Greenfield Sluder, St. Louis: The entire text of Dr. Grayson's paper is so at variance with the work I have done, beginning in 1909, that I feel impelled to recall some of the things that are within this discussion. I realized then, and I do now, that there are many elements, all of which Dr. Grayson had cited, which must be considered. Patients are sometimes highly neurotic and neurasthenic, and all of the various features he has mentioned are there, but if you can trace some of these patients back to the beginning, you will find that up to a certain time they were in good health and supposed to be normal; that they began with a violent coryza, with a terrific headache, and in the wake of that followed the pain in their neck, chest, etc., conceived by the neurologist as pathognomonic of neurasthenia. The pain in their necks and stiff necks was transferred to a "dislocated vertebra," and for twenty years they were spoken of as neurasthenic. Watching these cases from time to time, it has been my opportunity to make out that preceding these symptoms was a sphenoidal postempyema, and in the wake of that remained a subacute inflammation, sometimes localized sharply to the sphenoid palatin formation or the membrane which overlies the nasal ganglion. The membrane overlying the nasal ganglion is a sharply inflamed patch of redness, clear as one of the lamps on the wall, and an application of cocain to that particular point will relieve all the symptoms—the pain in the neck will vanish, the eyes become normal, etc.

It seems very difficult to work out that such a case in an able-bodied normal man, hard worked and responsible in life in every way, should point to the fact that that individual had suddenly become neurotic.

I shall not say anything more about it other than that my observations are in direct diametric variance with those of Dr. Grayson.

Dr. Burt R. Shurly, Detroit: It seems to me that we all err in taking care of this class of cases because of the fact that we do not classify them where they properly belong. In my opinion these cases should at least come under three large classifications: (1) Those cases which are sensitized to some particular condition, a food sensitization perhaps; (2) a so-called pollen sensitization; (3) a sensitization to some other chemic phenomenon.

We are just beginning to understand a few of the phenomena which may give rise to these devious motor disturbances. When we carry this subject deeper and farther, I think that these three classifications will include those cases which Dr. Sluder has mentioned, where we have termination in a purely localized condition; again, where we have a sensitization; and still again, where we have what has been so beautifully demonstrated in the laboratory with the carrier pigeon—a condition of early nerve fatigue in the granules in the giant cells of the spinal cord. You can take a carrier pigeon that has been on a long flight and examine histologically the giant cells of the spinal cord and find it completely changed from the picture of another pigeon's cells histologically. The granules in the first pigeon are absolutely used up, and the whole problem of nerve fatigue following that long flight of the carrier pigeon can be absolutely seen under the microscope. It is very remarkable—the change which the nerve cells adopt.

It would, therefore, seem very easy for that class of neurasthenics whose vasomotor function is less under hereditary disability, to have nerve fatigue develop under the slightest exercise. It seems to me that we must be very much broader in our consideration of this subject.

Dr. George A. Leland, Boston: It had seemed to me for a long time that we have an autointoxication in these cases. Why are some patients so highly susceptible to nascent proteins? Is it not because they are so hypersensitive to all proteins? That means that we are overfed in a nitrogenous way. If a patient is highly sensitized to nitrogen, if he has autointoxication from hyperacidity of his blood, shown by the hyperacidity of the urine, then it is reasonable to suppose that a nascent proteid in its most terrible state coming from a germ of pollen should set up these nasal neuroses. I do not consider it altogether a case of neurasthenia, but the "last straw breaks the camel's back." The patient is hypersensitized and therefore affected by these proteids.

He cited a case of a fashionable young lady, about thirty years of age, who came into his office one day, very indignant because she had been treated for a long time in various ways without obtaining any relief. So he asked about her general condition, and in the course of the questioning it developed that previous to four years before she saw him, she had been a stenographer, living very simply in a cheap boarding house and taking a normal amount of exercise each day. For the past four years she had been married to a wealthy man, and since that time had been living at a very fashionable hotel in Boston, eating heavy and rich foods, drinking regularly, and not exercising at all. When she had finished the story I told her, "Back to the simple life for yours!" and put her on a proper diet, and in less than three months she had no more hay fever and has had no more since.

Dr. Lewis A. Coffin, New York City: I have had some satisfaction in my treatment of this class of cases, which has been direct general treatment of the patient, particularly in regard to diet, elimination and cleaning up of the bowel.

Dr. J. Solis Cohen, Philadelphia: It has given me great pleasure to hear the paper under discussion, because it confirms the opinion I expressed more than a generation ago in one word—"coddling."

Dr. Joseph L. Goodale, Boston: There is undoubtedly a psychic effect in some forms of this condition. We know that the outbreak of hay fever in certain cases frequently occurs on exactly the same day every year, notwithstanding the great seasonal variations and the fact that the pollen does not appear always on the first of August. There are certain cases in which the hay fever appears on the first of August regularly, and that is the result, it seems to me, of a psychic expectancy. The man who went into the barn to set the broken leg of his friend was not thinking about his hay fever, and that is another reason why it probably did not develop. His psychism was not fixed upon the fact that he might develop hay fever.

Dr. Charles P. Grayson, Philadelphia (closing the discussion): I did not intend to teach anything new in this paper. My only intention was to refer to and emphasize certain facts of comparatively recent discovery or elaboration. I think Dr. Shurly's classification of these different cases is a thing we ought to be particularly cognizant of in every new case we examine.

There is no question of the occurrence and existence of cases such as those referred to by Dr. Sluder, but they are in a class by themselves. If he means to imply that it is a very large and extensive class, I have my doubts. Of course, there are a great many in the aggregate, but the proportion to all others is, I think, comparatively small.

The Relations of the Sphenoid Sinus to the Eustachian Tube, and Their Possible Clinical Importance.

By GREENFIELD SLUDER, M. D.,

ST. LOUIS.

When the sphenoid cell is prolonged downward into the pterygoid process it approaches the eustachian tube more or less, and it may come to such close association as to be separated by an egg shell thickness of the bone. This comes through two factors: first, the thickness of the bone of the pterygoid process is absent, and second, the origin of the tensor palati is not from the uppermost limit of the plate. Should the tensor palati arise high on the plate, the thickness of the muscle will enter in between the tube and the bone of the sinus wall. Should, however, the tensor extend a lesser distance upward on the plate, the tube comes into close relation and in some specimens is in contact with the egg shell thick wall of the sinus.

The Relations of the Sphenoid Sinus to the Semilunar (Gasserian) Ganglion, and Their Possible Clinical Importance.

By GREENFIELD SLUDER, M. D.,

ST. LOUIS.

The semilunar ganglion is at present thought of as so far removed from the sphenoidal sinus that they have not been associated in the minds of the anatomists or clinicians. The internal carotid artery usually rises on the lateral aspect of the body of the sphenoid, and the semilunar ganglion is usually lateral and posterior to the ascending artery. This, however, is not always true. The position of the artery seems to have a large part in determining these relations.

I present drawings of two specimens showing an intimate association of the sphenoid sinus to the semilunar ganglion, or parts of it. Figure 1 shows dissection of the cavernous sinus, semilunar ganglion and sphenoidal fissure, viewed from above. The sphenoid sinus lying beneath these structures is shown in dotted line. The thickness of bone separating the cavity of the sphenoid from these structures was egg shell thin. The internal two-thirds of the ganglion are exposed to the uppermost part of the sphenoid cell, and the external third is exposed at a little greater depth as the nerve tissues approach the foramen rotundum. The mandibular nerve in the foramen rotundum is exposed for ten millimeters to an egg shell bone separation from the sphenoid cell.

In the sphenoidal fissure the oculomotor, abducens and ophthalmic are exposed to an egg shell separation from the sphenoid cell. The trochlearis alone is not in this contact, because it lies on top of the oculomotor.

In 1912 I proved the permeability of the sphenoid sinus wall to a small amount of cocain. Following this observation, with its lesson, into these anatomic associations seems to me to offer an explanation of the herpes which develop in the wake of sphenoidal infections, or the "cold sores" which arise in some patients from ordinary coryzas, irritation of the sensory ganglion cells being necessary for the development of herpes, and to explain why semilunar ganglion neuralgias and tic douloureaux of sphenoidal origin sometimes recover as a more or less acute or subacute lesion, and at other times requires a ganglion removal or a posterior root section—i. e., sometimes the sphenoid lesion can be controlled, and at other times it cannot.

A Surgical Consideration of the Upper Paranasal Cells.

By Greenfield Sluder, M. D.,

St. Louis.

The procedure I describe has been satisfactory in my hands for ten years. It may be limited for the frontal sinus, providing a very high cut of the middle turbinate, which I have frequently mentioned in various texts as a "cribriform or infundibular turbinectomy." The cut is actually two or two and one-half millimeters from the cribriform plate, and may be extended to the most anterior limit of the infundibulum.

This very high cut may be carried backward to include the capsule of the ethmoid, under which condition not only is the middle turbinate removed, but the uppermost line, and usually all the other ethmoidal cells are opened wide into the nasal cavity. And when desirable the entire anterior wall of the body of the sphenoid from its uppermost limits, with all its postethmoidal association and much of its floor, may be removed. In my judgment and experience it is the technic that most often may be trusted to open all of the cells, regardless of unusual or anomalous positions. I know, however, that cells may exist that not only cannot be opened from the nose, but cannot be located by any means at present at our command. They can only be recognized in the cadaver, and then only by complete dissection. Everyone who has observed a reasonable number of specimens has seen such cells and been struck by the futility that would attend any surgical effort to reach them; and everyone of reasonably wide experience has met cases where all efforts had failed to find and treat such cells. But there are many other cells that are placed in positions more or less unusual that will be opened by a technic which has for its primary plan an incision which will skirt the cribriform plate and remove the middle turbinate at its most anterior as well as its most upper limits, and extend into the sphenoid body at its uppermost part, regardless of the natural opening, and then be extended downward until it has cut through its floor or found it to be impenetrable. I have here avoided the designation of sphenoid sinus, because such a term does not always comprehend the full body of the sphenoid. It (the body) may be subdivided, and shared by a postethmoidal cell. And it is this variation which I believe to be the most pernicious from the clinical side (for headache and optic nerve lesions), as well as anatomically far more frequent than is at present recognized clinically. This variation will, it seems to me, be satisfactorily dealt with by this proposed technic practically every time it is met. Also prefrontal ethmoidal cells will be opened into the nose as a rule, if not uniformly.

In 1907 I published an elementary text in which I described a surgical method which was at that time novel. It consisted in approaching the turbinate from above on its inner side. Prior to that all surgical approach was from below laterally upward, removing it by scissors or snare, or a combination of these or such working instruments. I described a knife consisting of a handle, a shaft and a cutting end turned at a right angle to the shaft and sharpened so as to cut on the inside of the right angle—i. e., on the pull. It was also sharpened on a face parallel to the shaft, which at the same time gave it far more strength than a hook could have were it sharpened on its concavity. I selected this shape in preference to a hook for those reasons, and the fact that great strength is necessary for the tasks to which this knife is put. The knife, although possessing great strength, is so small that it may readily enter spaces which larger instruments could not. I believe it to be the smallest nasal instrument possessing great strength.

The intranasal surgery of the upper cells may be performed by this method in any part or the whole as conservatively or as radically as desired. The ability to place the incision safely two millimeters below the cribriform plate in any part of or in the whole length of its extent seems to me to be the most advantageous, and not a small part of this advantage is the power to extend this incision to the foremost limit of the infundibulum, thereby opening the inlet of the frontal to its widest natural possibilities. It is most desirable to preserve the natural inlet here, and this is done by a cribriform turbinectomy which leaves undisturbed the histologic epithelial covering of the normal inlet—i. e., the uncinate process, the bulla ethmoidalis, the hiatus semilunaris and infundibulum—regardless of the anatomic variations of the frontal inlet. Should these parts be wounded, as in a curettement, the resultant scar tissue blocks the inlet. The angle knife removes by cutting any desired tissue with the least possible trauma to the surroundings. In the sphenoidal district it opens the uppermost and lowermost possible parts of the face, which has the advantage sometimes of opening also a postethmoidal cell which may occupy part of the body of the sphenoid. (Such a cell is often the cause of the entire clinical picture.) The angle knife is so small that it takes up the minimum room, and so leaves the small field open to the best vision possible. Its execution is always in the direction away from the danger zone. I have so far not seen such satisfactory postethmoidal surgery by other methods. (This district seems to me the most dangerous of all, for on the outer upper aspect runs the optic chiasm and cranial cavity.) Satisfactory execution is necessary, particularly in eye lesions.

This entire performance may be accomplished within a short time. I have often finished the high frontal ethmoidal and sphenoidal combined operation in two minutes (including the postorbital opening on one occasion).

A septum nasi deflected into the affected side may add troubles for the surgeon. I have in such cases where the middle turbinate was not visible from in front in any part, used a bivalve speculum specially constructed for the purpose. Just as Killian elongated the blades of the primary bivalve for his needs in septum resection, I have elongated them still more and widened them for my needs here. The speculum should be made of tempered steel blades, knife-like thin, with handles long enough to give leverage to dislocate the entire septum into the opposite nostril. It should have a set screw, for the pressure required is much, and the hand gets tired holding it. Its blades should be nine millimeters wide, for narrower ones, such as are supplied with Killian's, often do not give sufficient view. They should be eighty-eight centimeters long, for shorter ones (Killian's) sometimes fail to reach the posterior field of operation.

The knives are also made right and left. Some of my associates prefer these. My own preference is for the single, straight, original model. I recommend an extra two and one-half centimeters long shaft for use with the long blade sphenoidal speculum. A strong, large handle ought always be supplied for the knife.

A right-hand surgeon makes his turbinate cuts for the left side with cutting edges in the plane of the handle. For the right side the cutting edges are turned one-quarter (ninety degrees) into right nostril. This makes them horizontal or transverse when the handle is on the middle line.

DISCUSSION.

Dr. Harris P. Mosher, Boston: I feel that in working on the ethmoid the questions of time and an accurate knowledge of the anatomy, with a simplification of all procedures are essentials. If you can come down to two minutes, it is about as simple as you can make any procedure on the ethmoid. With Dr. Sluder I think two minutes would be a long time for him in which to do the operation. Again, the fewer instruments you can get along with, the better you are off. I have never done much with Dr. Sluder's knife, but I have spoken to a number of men who have used it and liked it. I believe that for work on the ethmoid we ought to come down to two or three instruments—instruments which are large enough to see and easy to follow from the beginning of their use. For instance, I use a simple straight curette, and the other ordinary instruments.

My feeling is that simplification of the procedure, simple instruments, and an accurate knowledge of the anatomy are essentials in dealing with the ethmoid. Anything which helps that along is welcome; anything which adds to the complexity of the instruments adds to the difficulties.

Dr. J. Winslow, Balitmore: I find the Sluder knife of the greatest use in practically all nasal sinuses, and employ practically no other instruments in work on the middle turbinate, unless it be a pair of tiny forceps or snare. One reason that attracted me to the instruments in the beginning was that I had a number of cases with suppuration and deviation of the septum, and it was extremely desirable that there should be good drainage. At that time I did not feel it proper to perform the deviated septum operation, and required some instrument small enough to correct the condition. These knives served the purpose.

Dr. Christian R. Holmes, Cincinnati: I want to congratulate Dr. Sluder upon his most excellent paper. I think, however, that it is well enough for us to bear in mind that we are in a most dangerous region, and what can be done by Dr. Sluder, an expert, cannot be done safely by inexperienced hands. We are in a region where we operate because there is infection, as a rule.

I have in mind a case where I wanted to do a Killian, cleaning out all the cavities. It really was a case of ozena, but I attributed it to disease of the sinuses and wanted to remove the middle turbinate before proceeding with the general operation, both for the purpose of testing the condition, and seeing whether there was a tendency to active suppuration, etc. The removal was very carefully done, under all precautions, and in spite of that the patient developed meningitis and died. And here the middle turbinate was removed by scissors only, so there was no special trauma.

I am not in any way decrying the method as presented, but am simply calling attention to the fact that we send out these articles without a word of warning to the inexperienced men who read them and learn about these instruments without ever having had an opportunity of using them. This is very dangerous, and I think we are not doing our full duty. It seems to me that Dr. Sluder has not sufficiently emphasized the fact that there is danger, unless we use special care, and that inexperienced men, who do not know the anatomy or bacteriology, cannot consider that they can buy the instrument without knowing how to use it, and then begin to work around the cribriform plate.

Dr. Greenfield Sluder, St. Louis (closing the discussion): Had I adhered to my text, Dr. Holmes would have been answered. It is emphasized that the district must be approached with care and understanding.

I fancy that Dr. Mosher's idea of the simplification of instruments is not quite right, from the number that I have turned in to the chair. There is one knife which may be used for the entire procedure, if you stop to turn it this way and that. If you want to work faster, you can have three knives turning in various directions. Others are unnecessary except for more or less refinement in technic.

I think, however, that the only reason I have for appearing before you today with this technic is that it affords a method which cuts everything downward and outward. If there is any device which can make surgery at or approximating the cribriform place safe, it is in our hands, and it seems to me that whosoever will approach the district with this method shall have a means which will let him get his bearings, feel the cribriform plate, and then begin his cut. If the knife is big enough, it can tear into the orbit, and that is the reason I have emphasized the necessity of the surgeon knowing his model.

I am glad Dr. Holmes emphasized the necessity of care being taken and emphasis being laid upon who shall and who shall not work in this region. I am perfectly aware and alive to the fact that there are many surgeons who buy instruments from the instrument maker and do not familiarize themselves either with the method which requires the instrument or even the directions accompanying the instrument. There is no help for that; we cannot appeal to such a man nor emphasize the necessity of his knowing just what he is doing. He must know his anatomy, first, last and forever; and secondly, he must have an instrument which will then make his procedure as safe as the procedure may be made.

The Tonsilloscope and the Exploration of the Interior of the Tonsils in Situ.

By Thomas R. French, M. D.,

Brooklyn.

The speaker has devised a method by which the external tonsilloscope, originally intended and used for the examinations of exploratory sections removed from the tonsil at the beginning of operations, and now used for study of the tonsil, as a whole or in part, after operations, may be used for direct tonsilloscopy or for the examination of the tonsil in situ. The introduction of the instrument into the throat is accomplished by using the shortest of the Jackson bronchoscopes with a beveled end and a lamp on a light carrier of the next largest tube, packed securely with gauze so that the lamp is held just within the distal opening and the end of the tube slipped behind the tonsil. In this way the tonsil is lighted up as brilliantly as in the powerful external apparatus. With this instrument the writer has studied the various classes of tonsillar disease, including three hundred and thirty-three cases that were operated upon, and a large but indefinite number of youths and adults. He concludes that while this may not permit definite and final deductions, nevertheless the results seem to indicate that the erstwhile enlarged tonsils of health in childhood may be regarded as permanently diseased if they continue enlarged after the seventh or eighth year, and that the enlarged tonsils of health which undergo a retrograde metamorphosis in late childhood may remain the tonsils of health throughout life. The assumption that the tonsil has no special function because it has not yet been discovered, is an unconscious confession of impotence to which few could agree, but one conclusion reached in this study is that the function of the tonsil is a negligible feature, for it must be conceded that a tonsil which is extensively diseased would probably be the potential or actual source of too much mischief to be offset by the value of any function which a part of it might possess. When, however, a tonsil is found to be that of health, it should be left at least in part to perform whatever function it may have, and also incidentally to spare the fauces the now common postoperative deformities and the consequent impairment of the speaking and singing voices.

DISCUSSION.

DR. D. BRYSON DELAVAN, New York City: This work is very real in its simplicity and its practicability, and very real in the ease in which it can be applied; there is nothing in it which would not come within the technic skill of any ordinary operator. It is all that you think it to be from the short presentation of it, and a great deal more besides.

DR. E. FLETCHER INGALS, Chicago: I would just like to ask Dr. French how large a tonsil must be before he calls it "enlarged."

DR. HENRY L. SWAIN, New Haven: When I wrote a paper some time ago in defense of the much slaughtered tonsil, my whole thought was that we ought to be able to tell in young children, under age, whether a tonsil was diseased or not. I want to congratulate Dr. French, for I think it is a very wonderful thing.

DR. CHEVALIER JACKSON, Pittsburgh: Long ago I gave up the tonsil as a "bad job"—one not for me to do. It requires more skill than I could bring to bear upon the subject. I have, however, a distinct recollection of talking various cases over with members of this society, and I find that there is a very fluctuating idea as to what constitutes a diseased tonsil, apart from mere enlargement. It seems to me that we are arriving at a stage of accurate diagnosis when we study the tonsil in this way.

Dr. John F. Barnhill, Indianapolis: On two occasions last year I remember tonsil patients were brought to my observation, and in neither case could I find any trouble. I was frank to say so, and stated I did not think the tonsils should be removed. In each instance the family physician said: "There is some condition here which I am confident has arisen in the tonsil," and upon that statement and his recommendatoin, I was willing to do the work. In each case I found a cold abscess outside of the capsule.

I am wondering if Dr. French's method would have been able to discern this. In each instance the abscess had been present for at least a year.

Dr. Hanau W. Loeb, St. Louis: There are two points I would like to know: If the instruments can be procured, and where; and secondly, whether or not the method will demonstrate the presence of the minute multiple foci of pus in various portions of the tonsil.

Dr. Greenfield Sluder, St. Louis: These are the observations for which Dr. French's friends have been waiting so impatiently. I have known of what he was trying to do and something of how far he was succeeding.

Dr. Thomas R. French, Brooklyn (closing the discussion): In answer to Dr. Ingal's question as to the definition of the word "enlarged," that was covered in the part of the paper not read. I tried to make it clear that I referred and do refer in such tonsils to glands which project beyond the edges of the anterior pillars, or sufficiently large to curve the anterior pillar forwards.

In answer to Dr. Loeb, I don't know who makes the instruments; these were made for me by Hardy, at Twenty-second street, New York, and I presume he would make them if wanted.

Without Dr. Sluder's assistance and his brilliant technic in the first stage of the experiments, I do not think this thing could have been accomplished. Without the first stage there would not have been any second or final stage, and I am under great obligations to Dr. Sluder for that work.

As to the ease of finding, perhaps a moment might be well spent in listening to the description of a patient who came under my care about six weeks ago, and was under my care for two weeks. She came to me for labyrinthine deafness on one side. For eighteen months previous to the time I saw her, her hearing had been absolutely blank and responded to nothing. The patient was a school teacher, and a very intelligent lady. She had read Dr. Shambaugh's article on infection, in which he suggests rather than justifies the infection. I examined her and found she had a tonsil which would have made a hypertrophied tonsil. Yet with this method I saw four different collections in that tonsil, and on the basis of that, and with continual reference to the article as to the thing I was doing, took out one; at the end of the second week I got out the fourth one, and she came in the following day with her face like the sky in the morning. Her hearing had entirely returned and absolutely responded to every test as completely normal. It was very easy to find those; with the aid of a lamp I got out the fourth one. Yet her tonsil is still hypertrophic, and along with these areas there is a mild general hyperemia which indicates the presence of at least a possible irritating substance. It really was one of the most startling cases, and indicates what must of necessity come to us at times.

Endothelioma of the Right Bronchus Removed by Peroral Bronchoscopy.

By Chevalier Jackson, M. D.,

Pittsburgh.

A man of thirty-five years had complained of wheezing, cough and mucopurulent expectoration of five years' duration. For two years he had been treated for tuberculosis, though bacilli had never been found in the sputum. Diagnosis of various clinicians had been: tuberculosis, chronic right-sided bronchitis, monolateral (right asthma). Dr. J. C. DaCosta had made a correct diagnosis of stenosis of the right bronchus. Radiograph showed dense shadow of lower right lobe, and in consultation diagnostic bronchoscopy was urged.

Dr. Jackson found and removed bronchoscopically a pedunculated tumor which had made for itself a dilatation in the right bronchus. Patient entirely recovered in two weeks, expectoration disappeared completely at the end of four weeks.

At the end of ten months patient had gained twenty-six pounds in weight and was perfectly well. The pathologist reported the growth to be an endothelioma. The author makes the following conclusions:

1. Diagnostic bronchoscopy is indicated in cases of monolateral "asthma," bronchitis, bronchial obstruction, and in cases of tuberculosis where persistent search fails to show tubercle bacilli.

2. Peroral bronchoscopic removal of an endobronchial tumor is feasible under local anesthesia.

3. General anesthesia might have permitted the clotting of blood in the lower bronchi before expulsion, involving septic risk.

4. Peroral bronchoscopic removal may be justifiable in a malignant endobronchial growth if small, circumscribed and not ulcerated.

5. As this is the only recorded case of apparent cure of an endothelial endobronchial tumor by peroral bronchoscopy, and only the second endoscopic removal of any form of malignant growth from a bronchus, it would be unwise to make too many or too sweeping deductions.

Report of the Removal of a Fragment of a Tracheotomy Tube From the Lung, Six Years After Its Inspiration.

By Francis R. Packard, M. D.,

Philadelphia.

An Italian, thirty-three years old. Twelve years previous to present complaint had typhoid fever, necessitating intubation. On removal of the intubation tube had tracheotomy performed, and has worn the tracheotomy tube ever since. The past six years had suffered from violent attacks of coughing, accompanied by expectoration of blood and much pain in his chest. An X-ray examination revealed a foreign body lodged in the right bronchus opposite the left intercostal space. The patient was etherized and the foreign body removed by means of the Jackson bronchoscope. The symptoms cleared up and the patient refused to allow the tracheotomy wound to be closed, although it seems as though this might have been done and his proper method of breathing restored to him.

DISCUSSION.

DR. J. SOLIS COHEN, Philadelphia: A great many of the tracheotomy patients keep their tubes in without taking them out for cleansing, and in the course of time they become more or less detached. I have tried to prevent this by having my tubes made without the solder. I then adopted the plan which I have recorded in my reports of always insisting upon a patient having two sets of tubes, and changing them on alternating days. In that way you will avoid any instance of this kind. The patient can take it out and cleanse it and put a new fresh tube in.

A Case of Vagotonia, Apparently Originating in the Nasal Accessory Sinuses.

BY GEORGE FETTEROLF, M. D.,

PHILADELPHIA.

Vagotonia can be defined briefly as a condition of excitement or high tonus of a group of nerves called the "extended vagus." The phrase "extended vagus" is applied to a nerve series which includes not only the pneumogastric, but also a group of nerves which functionate similarly to it. Antagonistic to the vagus group is the sympathetic, and there exists a condition of it called sympatheticotonia, which can be defined as a state of excitement of high tonus of the sympathetic system. In order to locate accurately the "extended vagus" and sympathetic systems, the writer reviews briefly some points in the anatomy of the nervous system.

In action the two groups, the autonomic and the sympathetic, are antagonistic, and it is generally conceded that all glands which possess ducts and all involuntary musculature receive a supply from both. Upon the maintenance of a proper balance between the two depends the normal functionating of the structures to which they go. If the autonomic supply is irritable, the organ will overfunctionate in one direction, and if the sympathetic is in a condition of hypertonus or excitement, the excess of action will be in the other.

As glands and muscles are the organs to which these impulses go, the result will be manifested by hyper- or hyposecretion on the one hand, or by spasm or relaxation on the other.

The control of the entire vegetative system, both autonomic and sympathetic, is believed to lie in the cerebrospinal axis, where possibly a regulating center exists, and in the glands of internal secretion, the socalled endocrinous glands. This latter is quite well established for the sympathetic system, adrenin having been shown to act generally as a stimulator to the entire distribution of this group. For the autonomic system no drug has been found which acts uniformly upon all parts of it. The nearest approach to such a drug is atropin, which is a sedative to practically the entire autonomic system, dilating the pupil, checking the flow of saliva and sweat, and relaxing contracted involuntary muscle.

Even more selective is pilocarpin, which has a powerful effect upon the salivary and sweat glands, producing oversecretion, and hence clearly to be regarded as a stimulator of part of this system.

Pilocarpin is known to be so reliable and constant a stimulator of the autonomic system that it is used in testing for the presence of vagotonia. One-twentieth to one-sixth grain is given hypodermically, and if the test is positive there will be noted salivation, lacrimation, sweating, cardiorespiratory, erythema and hyperperistalsis, all these out of proportion to the size of the dose.

The symptoms of vagotonia in general are such as would be expected when the autonomic system is stimulated, and they represent activity of function at the terminal distribution of the nerves concerned.

There are many of these symptoms, but just a few are mentioned to indicate their general nature.

Cramp of the ciliary muscle, spasm of accommodation and widening of the palpebral fissure are examples of what would result from hypertonus of the fibers from the ciliary ganglion.

Salivation and congestion of and hypersecretion from the nose and nasopharynx would follow irritation transmitted through the otic, sphenopalatin and submaxillary ganglia.

To the vagus itself can be ascribed many phenomena, such as bronchial asthma, bronchial hypersecretion, laryngeal crises, bradycardia, gastric crises, hyperperistalsis and excessive gastric and intestinal secretion.

Through the sacral part of the autonomic system could pass impulses which would give rise to such conditions as spasm of the anal sphincter, hypersecretion from the intestine, dysmenorrhea, etc.

The blood picture shows an excess of eosinophiles.

The treatment of the condition is the same as that of any other secondary disease—viz., find if possible and eliminate a cause or focus, build up the health and give proper medication. The last factor, the medicinal treatment, can be managed on one of two principles, either administering a sedative for the autonomic system, or overcoming the tonus of the latter by giving a stimulator to its antagonist, the sympathetic system. This principle is illustrated notably by the successful treatment of spasmodic bronchial asthma, success being possible either by quieting the autonomic system with atropin or by stimulating the sympathetic by the local or hypodermic use of adrenin.

The writer cites a case in point:

A boy of twelve years, who had failed in health for five years and had an intermission or two of slight improvement. He presented many symptoms of autonomic tonus, and, in addition, had evident disease of his paranasal sinuses.

His impaired health dated back to an attack of measles at the age of seven, which left him with marked nasal discharge and expectoration, which have persisted ever since. There was at times a loose cough, which was productive of a thin, mucoid sputum, which never became yellow nor blood tinged. For a year there was profuse sweating at night, and his sleep was much disturbed by sneezing, coughing and expectorating. His adenoids were removed during this time, but the result was unfortunate, as immediately afterward his nasal discharge increased and he developed abscesses in both ears.

Three years after the appearance of the first symptoms, Dr. Stengel saw him. No cause for his symptoms was found, physical examination being negative. He was tested with pilocarpin for vagotonia by Dr. Hopkins, and the result was positive. He was put upon guaiacol carbonat and extract of belladonna, and some slight improvement resulted.

Two years later he returned to Dr. Stengel, a very sick little boy, having lost nine pounds in four weeks. At this time he was vomiting all his meals, a condition which soon changed to a weekly attack of severe abdominal pain, nausea and vomiting, which would last about two days. The vomitus was profuse and consisted almost entirely of mucus. With this there was profuse salivation, of a clear, watery character. Soon was added to these conditions frequent watery bowel movements. Medication was of little value, and the boy's condition was becoming a desperate one.

At this time the writer was requested to examine him, and aided by an X-ray examination a diagnosis of chronic suppuration in the posterior ethmoidal and sphenoidal cells of the left side was readily made. The surgical indications were clear, and he removed the posterior end of the middle turbinate and opened widely the offending cells.

Improvement was immediate and very striking, as almost at once his symptoms disappeared. In two months he gained ten pounds, and was a perfectly well boy except for some discharge from his nose. Later autogenous vaccines were given, and this was followed by almost total disappearance of the nasal mucus.

The interpretation of the case is this: The boy was one with a very unstable vegetative nervous system, and it has been observed that in such a state small stimuli can produce large reactions. The irritation which would cause such a condition of autonomic tonus may "arise from noxa in the form of bacterial toxins, as during or after acute infections, from drugs, or from the products of deranged metabolism, mechanical irritation, and so forth" (Hopkins).

There was present in this case a primary acute infection, measles, followed by prolonged chronic suppurative sinus disease, and we believe that the consequent toxemia and irritation were the exciting factors. For, when this was relieved, the widespread vagotonic symptoms disappeared so promptly as practically to bar accidental coincidence.

As far as the writer has been able to discover, the case is the first on record in which such a distinct entity as chronic sinus disease was presumably the exciting cause of such widespread vagotonic manifestations, and in which prompt disappearance of these symptoms followed relief of the local condition. By the free drainage of suppurating sinuses this patient was almost instantaneously relieved of salivation, cough, expectoration, severe abdominal pain, vomiting and diarrhea, for none of which symptoms a physical cause could be found.

As a sinus case per se there was nothing notable about it, as the diagnosis was easy and the treatment simple. The big feature is the field of observation and deduction such a case opens up. It does seem that many other conditions which are but vaguely understood may possibly become clarified when viewed as vagotonic or sympatheticotonic phenomena. For example: The treatment of bronchial asthma with adrenin or with atropin has been successful in many instances.

Noteworthy has been the nasal treatment by our colleague, Emil Mayer, Brettauer and others, of selected cases of dysmenorrhea.

Another fellow member, Greenfield Sluder, after painstaking study, has succeeded in relieving certain symptoms by treating the sphenopalatin ganglion.

These are but few of the conditions upon which some light may be shed. Undoubtedly, many others will suggest themselves as being based upon a derangement of visceral nerve supply. Should such prove to be the case, they will constitute a new group whose pathology can be described in definite terms.

DISCUSSION.

DR. G. HUDSON MAKUEN, Philadelphia: This most timely paper by Dr. Fetterolf is exceedingly interesting to me because it expresses very well and emphasizes again the close relationship which exists between our own specialty and that of neurology and general medicine.

DR. J. GORDON WILSON, Chicago: The advantages offered by the supporters of the vagotonic hypothesis are that it offers a plausible interpretation and suggests a rational treatment of certain types of neurosis. As it deals so largely with the vagus nerve, the nerve which innervates so many of the parts we specialize in, it is incumbent on us to learn on what basis this hypothesis rests.

Eppinger and Hess, whose statement is authoritative, state that "vagotonia is a functional increase of tone in the autonomic system, which permits stimuli to act more readily on it," and regard it as a state of stimulation of the vagus. The vagotonic disposition is abnormal irritability. "Its cause is to be sought in disturbance of internal secretion, and it is present if we have increased sensitivity to pilocarpin."

If vagotonia is an increase of tonus in the autonomic system, it will be well here to inquire what is the conception of tonus that is usually accepted. Tonus is a difficult thing to define —it seems at times so intangible. There are three sets of phenomena to which physiologists usually apply this term.

1. Prolonged contraction of smooth muscle which is automatically independent of impulses from nerve centers.
2. A state of tension shown by skeletal muscles under certain conditions which in some way is dependent on nerve stimuli, for it disappears if these be cut.
3. State of some nerve centers which appear to give off constantly nerve impulses apart from the receipt of messages from other nerves—e. g., the respiratory nerve center.

It is with the last of these that we are chiefly concerned.

Dr. Fetterolf has sufficiently referred to the anatomy of the sympathetic nervous system ("vegetative system") and its two divisions, the autonomic nervous system (vagus group), and sympathetic nervous system proper. This division has been long recognized and acknowledged chiefly through the work of the English physiologist, Langley.

I do not intend to discuss whether or not the supporters of this theory have sufficiently differentiated between stimulation of a nerve and nerve tonus. I wish, rather, to call your attention to what is one of the fundamental propositions on which this vagotonic hypothesis is built. Eppinger and Hess say that tonus in the vegetative system is due to the equilibrium of two impulses, one from the autonomic and one from the sympathetic system, caused by two hormones antagonistic to each other. Hormones, as you are aware, are chemical messengers by means of which the activity of certain organs is coordinated with that of others; produced in one organ, they are carried to another, which they affect, even when present in very small amount. The supporters of the vagotonic theory assume as a fundamental fact that sympathetic tonus is produced by adrenalin, and to get a corresponding hormone for the autonomic system, they further assume, without any evidence, the existence of an unknown hormone akin to pilocarpin. According to them, these two hormones, the real and the hypothetic, acting in antagonism to each other, produce normally a state of equilibrium. A departure from this state of equilibrium constitutes in the autonomic system vagotonia.

The first point I would make is that in their attempt to establish the equilibrium essential to their hypothesis, they assume as proved beyond doubt that adrenalin acts to produce tonus. Most recent investigations appear to show that adrenalin does not produce tonus in the sympathetic system.

To get over the difficulty of the assumption of an unknown hormone to act on the autonomic system, they assume that this hypothetic hormone will act like pilocarpin. Pilocarpin is a chemical body which acts directly on the cell membrane and produces morphologic changes in the cell. It seems to me that there are serious objections to a hypothesis which demands that we grant to it for a basis an unknown hormone comparable in action to a chemical body. It would, therefore, appear that the fundamental basis on which this hypothesis is built is not generally accepted, and is open to very serious objections.

Again, it is assumed that the glands of internal secretion, the endocrine glands, control the sympathetic system, and that they produce and stimulate the tonus of the vegetative system. This appears to me to be an assumption beyond the point we are justified in going. Experiments with these glands, such as feeding with these glands and removal of these glands (e. g., pituitary and thyroid), show that their action is not so restricted, but that they affect the growth of many of the body cells. It would be more correct to say that it is generally accepted that the endocrine glands act on tissue metabolism, and so indirectly affect the condition of the nerve cell.

There might be quoted other examples of the unreliability or doubtfulness of the assumptions demanded by this hypothesis. Equally unreliable appear to be some of the applications of the hypothesis. It is stated (Eppinger and Hess) that "the stomach dilates actively in proportion to the degree of filling, thus causing the muscular elements to cover the contents closely. Tonus is, therefore, a resistance to filling." The meaning here is not quite clear. The two consecutive sentences do not fit in unless one lays emphasis on the "resistance to filling." The meaning appears to be that as the filling increases so does the tonus. We find that experimental evidence does not bear out the statement that tonus varies in proportion to the degree of filling. Tonus can only be measured by the tension of the muscle, and Sherrington has shown that the muscle wall of the living stomach can increase from an one hundred cubic centimeter posture to a five hundred cubic centimeter posture without change of tension. Also the reverse of this holds, for there may be loss of three hundred cubic centimeters without fall of pressure.

Vagotonia is a difficult subject to discuss, because it is based on the physiology of the sympathetic system and of the endocrine glands, much of which is unknown and much of which is speculative. The subject of the action of the sympathetic system in health and disease is a very important one, and if "vagotonia" has directed the attention of the clinician to this very important subject, it has served an excellent purpose. When from experience we find that atropin or pilocarpin is of use in diseases associated with the sympathetic or internal secretive system, we are justified in using them, but to build a system of medicine on so slim a basis as the clinical use of drugs is to court disaster.

In concluding, let me quote the words of Elliott, one of the leading investigators of the glands of internal secretion: "Medicine owes no debt of gratitude to those who teach to her theories without proof."

Dr. Thomas Hubbard, Toledo: I am surprised Dr. Fetterolf did not include esophageal spasm in the same category as bronchial spasm. I would include also the globus hystericus, the various types of esophagismus, and also cardiac spasm.

Dr. Emil Mayer, New York City: It seems to me that we have with all this entirely wandered from the question of vagotonia pure and simple. We have to explain some of the things we have not understood, as, for instance, the effect in treating for dysmenorrhea, as mentioned by the speaker. Above all, the immensely practical value in that is to always remember that when we have done anything surgical for our patients, our efforts must cease, we must not be surprised if the patient does not get well, and we must not put them down as being nasal hypochondriacs.

I have a case in mind of a gentleman who came to consult me recently. He had been having an ethmoiditis. The operation on his nose had been very properly and carefully done, but he was still having terrific headaches. He was one of those men who was determined to find out what the trouble was, and he was sent to me. I told him that so far as the nose was concerned, there was nothing to do for him surgically. He said that he was still having the discharge and still having the headaches. A vaccin of his secretion had been made, and he had received injections, but nothing had helped him. I noticed his tongue was very much furrowed, and inquired very carefully into the question as to whether he had syphilis. He said, "No"; he had absolutely no knowledge of it. A Wassermann test was made which proved negative. He even brought his sister to me, and she submitted to a most careful examination, and there was nothing found. I sent him to a dermatologist, and he said it was probably a case of hereditary syphilis, and he advised salvarsan and mercurial injections. The patient has now become entirely well, and as soon as he has any symptoms, a mercurial injection always makes him well.

I quote this case because it seems to have a marked connection with the subject at hand, and to show that we must remember that a patient has something else besides nose and throat.

DR. JOSEPH L. GOODALE, Boston: In view of what we know takes place in a child who is weaned and that which takes place also in conditions of asthma and in other anaphylactic conditions, this may possibly be an indication of the underlying identity of these various conditions.

DR. G. HUDSON MAKUEN, Philadelphia: I am particularly pleased that Dr. Fetterolf should have brought this subject before the meeting, because I have long been of the opinion that we as laryngologists and otologists have been giving rather too much attention to the surgical side of our specialty, to the exclusion, or perhaps neglect, of the neurologic and medical side of it. I, therefore, think that this discussion is very timely and very important. I am of the opinion that in the near future our work will deal perhaps less with surgery and more with the general phase of the subject.

Dr. George Fetterolf, Philadelphia (closing the discussion): In answer to Dr. Wilson, I would like to quote the reply to what was said about four years ago by Dr. Meltzer with regard to a certain criticism that a subject was five per cent fact and ninety-five per cent theory, but that a working hypothesis is better than none.

In answer to Dr. Hubbard, I would say that I have been busy during the last two months cutting a sixty-page paper down to thirteen pages. There are a great many phases that I have not been able to touch upon, and a great many symptoms and signs that I did not have the space to take up.

If anyone is interested in this, the most recent things which have appeared are a brochure by Eppinger and Hess, translated by Jeliffe and Kraus, and an exceedingly able article by Barton, which appeared in the Transactions of the Association of American Physicians in 1905.

Removal of a Large Rhinolith, With Exhibition of Specimen.

By D. Braden Kyle, M. D.,

Philadelphia.

Patient, male, aged seventeen years. The right nostril was markedly obstructed by the septum, which was deflected and perforated by pressure from the body in the left nostril. This obstruction in the left nostril was found to be extremely hard, almost black in color, and covered by a great quantity of mucopurulent secretion of bad odor. This object was large enough to fill the entire naris; it forced the septum to the extreme right, extending into the antrum, and could be felt projecting backward into the nasopharynx. It was freely movable and not attached at any point—pocketed, as it were.

An operation was performed October 8, 1914, under ether anesthesia. Attempts to remove the obstructing body in its entirety failed, owing to its large size and to the fact that it bore the relation to the surrounding bony structures of a ball to its socket. It was necessary to use heavy rongeur forceps, by means of which it was possible to break off small fragments from the granite-like mass. About one-third of the body was removed in this manner, when the remaining portion was removed entirely with only a slight tear at the nasolabial angle. The nose was packed with iodoform gauze. The entire operation occupied about one hour, and considerable blood was lost. The patient was cyanotic and in shock when taken to the ward.

Twenty-four hours after operation a distinct pneumonic area had developed in the lower left lung, and the patient was removed to the medical ward for treatment of this condition, which cleared up in due time. During the four or five days following the operation there developed considerable emphysema of the cellular tissue at the root of the neck and below the clavicles. The face was markedly swollen and the eyes puffy. By the 17th of October a distinct abscess had developed at the inner canthus of the left eye. This was incised by Dr. Sweet, and found to communicate with the anterior ethmoid cells. This complication being relieved, and the lung having become clear, the patient was returned to the special ward on October 19th, where he made an uneventful recovery, and left the hospital on November 11, 1914.

DISCUSSION.

Dr. Harris F. Mosher, Boston: The objection brought out by the speaker that the case was septic and he did not feel like working the incision through the cheek, does not seem a valid one. You can get perfect drainage in this way. Had he gone after the tumor by making that incision, I feel that he could have seen what he was up to and stood a very good chance of getting his tumor out whole, which, of course, to any surgeon, is a certain amount of satisfaction.

Dr. D. Braden Kyle, Philadelphia (closing the discussion): I would like to say that this tumor weighed two hundred and fifty-four grains. It seems to me that if you can remove a mass without any serious trauma to the patient, you are highly justified, for the satisfaction of having the entire rhinolith, to subject the patient to a major operation. But in this case one could not possibly extend into the nasopharynx and not have trauma without doing a very extensive operation and splitting the whole upper jaw. I feel that in this case removing it in small portions was a much better surgical procedure than the one mentioned.

Sarcoma of the Nose—Report of a Fatal Case, With Metastases in the Cervical Glands and in the Brain.

By J. Payson Clark, M. D.,

Boston.

A female, sixty-four years old, noticed a swelling on the nose for four months. When first seen by the writer it was about the size of a small marble. For a month there had been a slight bloody discharge from the nose. There was a dark purplish tumor visible on the outer wall of the left nostril just above the vestibule. The growth was completely removed. The microscopic diagnosis was either carcinoma or sarcoma. In two months a gland was felt under the left side of the jaw. This was excised and proved to be a spindle cell sarcoma, thus determining the nature of the original growth. Seven weeks later a gland was palpable under the left sternomastoid muscle. Coley treatment was tried, without apparent success, for two months. At the end of this time a small recurrence was found in the nose and removed, and a month later a complete glandular dissection of the left side of the neck was successfully done. Three weeks later the patient developed mental symptoms, incoherent talk, convulsive attacks, motor aphasia and dysphagia. At times she was very drowsy, and then restless and irritable. She soon lost power of locomotion and control of sphincters. She became very comatose and hard to rouse, and ate practically nothing for the last ten days of life. An autopsy showed three metastases in the brain.

The most interesting features of this case are the metastases in the brain and cervical glands. The writer could find but eight reported cases of sarcoma of the nose in which metastases were said to have occurred. Six of these cases are somewhat doubtful, leaving only two (besides the case here reported) in which metastases occurred in the cervical glands, and only one other case in which they occurred in the brain.

DISCUSSION.

Dr. Robert C. Myles, New York City: The question of sarcoma of the nose and accessory sinuses is an interesting one to me because of the optimistic feeling I have with regard to it. A few years ago I had two cases of sarcoma in the frontal sinuses, and by a complete operation and removing the bone extensively and all the periosteum, I was able to get a good result. One of the cases had invaded the orbit. It is now more than seven years, and neither of them has recurred.

Twelve to fifteen years ago I reported a case where the whole of the nose was involved, in which everything was removed very deeply, and that has not recurred.

About two years ago I had a case of extensive carcinoma, which first appeared to be sarcoma, involving the external wall and ethmoid, in which everything was removed—the antrum freely taken away with extensive removal of the periosteum. That patient, to my surprise, has had no recurrence, and since that time has developed remarkably physically and in every way. So that I don't know what it is that causes such dread of these cases. Personally, I think the good results must be something more in relation to the periosteum than to the bone. If that is thoroughly removed, even if you have to leave the bone, as you do especially when one of the orbital plates is involved, there is still a lot of hope. I think we ought not to let these cases go on. I remember the case of a woman who had sarcoma of the septum, and for the reason that we did not remove her septum to the cribriform plate, she died of metastases in the brain. With that idea of conservatism we did not remove the whole septum, and I think it was a mistake.

DR. J. PAYSON CLARK, Boston (closing the discussion): Of course, we have all seen a great many cases of sarcoma which have gotten well after thorough removal, but in this case of mine I feel that there is no question but that the glands of the neck and metastases in the brain had already occurred before I operated. I am led to that view from the fact that, apart from the slight recurrence in the nose, there was nothing to suggest the probability of extension of the growth. This was not a direct extension of the growth to the brain, but the growth was carried through the lymphatics.

Brain Abscess From Chronic Suppuration of the Frontal Sinus.

By T. Passmore Berens, M. D.,

New York.

In a previous communication the writer reported a case of this affection, with a record of forty-nine other cases found in the literature.

Since then six other cases have been recorded, and are herewith briefly narrated, to which the following is now added:

A male, aged thirty years, was operated on January 16, 1913, under gas-ether anesthesia. Radical external frontal and sphenoidal operation and opening of the antrum of Highmore through the nasoantro wall. Slow convalescence.

In December, 1913, pus in the nose. Old wound reopened through the nose and washed out. This had to be repeated in July of the following year. In December of the next year recurrence of discharge, and on January 5, 1916, there was a large swelling of the cicatrix in the right frontal region and headache, and the old wound was opened under anesthesia. Much pus flowed from the wound, and also from the antrum, which was opened.

Granulations in the roof of the sinus hid a perforation which led to a cavity two and one-eighth inches from the roof of the sinus. This opening was gently enlarged, and a horse hair drain placed therein.

Finally, a soft rubber drain was inserted, and the discharge continued for five weeks. The wound was not allowed to heal for some weeks thereafter.

Culture from brain showed streptococcus hemolyticus in pure culture, and the same were found in blood culture in small numbers the day after the operation.

Recovery complete except that there was still considerable discharge of pus from the left nostril.

Operations on Tonsils and Adenoids in Diphtheria Carriers.

By Thomas Hubbard, M. D.,

Toledo.

Classification of diphtheria carriers: (1) Primary or healthy carriers. (2) Secondary carriers.

1. Have never had clinical diphtheria showing perfect immunity toward the diphtheria bacilli found in nose and throat.

2. Includes persons who have had clinical diphtheria one or more times, and in whose secretions diphtheria bacilli can be found twenty-one days or more after last attack.

Local treatment is very unsatisfactory, as most of these cases have enlarged tonsils and adenoids, and crypts cannot be reached by antiseptic applications. However, it should be added that certain cases must be treated in this manner until in condition for the radical surgical method, and occasionally it will be successful.

Thorough tonsillectomy and adenoidectomy are proving very efficient in cleaning up carriers.

In group one, operations—that is, tonsillectomy and adenoidectomy—can be undertaken with confidence that the person is perfectly immune. In spite of the fact that false membranes appear in tonsillar fossæ, these patients recover in ordinary period.

In group two it must be borne in mind that the immunity is not dependable. Each case must be studied carefully. Immunity test of Schick, made before the time of operation, will aid decision.

In all of these cases exhibiting transient immunity the question of antitoxin naturally comes up. Anaphylaxis must be taken into consideration, tests of sensitization, etc., and all precautions taken in administration of serum at such intervals.

The physical condition, together with the skin reaction tests, should decide whether or not the patient is in condition for operation.

Case Report.—Secondary type. A carrier for several months after second attack of diphtheria. Immunity very transient. Recovery in one week after operation, even though genuine diphtheritic pseudomembrane was extensive. No diphtheria bacilli after ten days.

DISCUSSION.

Dr. Stanton Friedberg (as guest—by request): This problem of carriers was called to our attention several years ago by the fact that we had patients who, on account of having positive cultures in their throats, were compelled to remain in the hospital for weeks, sometimes fifty to sixty days. As we only have a limited capacity (thirty to forty beds), this was a serious problem, for here we had clinically well patients occupying beds which we really needed. We experimented along various medical lines without any particular effect in some of the cases, and it was then that Kale was brought out in these cases. In spite of all the local treatment we found that there were a number of cases that would not clear up, but had to remain in the hospital forty or fifty days. In these cases I would urge nasal cultures as well as pharyngeal, for we frequently found that we had positive nasal cultures when there were no positive pharyngeal cultures.

A certain class of these cases will clear up under medicinal treatment. We have tried out vaccins without any particular effect. The feasibility of medical treatment in some of these cases we can illustrate in this way: We know that we find the bacilli in the depths of the crypt, where it is impossible to reach the condition. It is here also discovered in the epithelium, and that perhaps explains why we do not get any effect from local applications of medicine.

As to those cases which involve the nasopharynx and the nasal accessory sinuses, as Dr. Hubbard has mentioned, the accessory sinus cases are the hardest ones to deal with. To illustrate the futility of the use of the staphylococcus spray, I would cite a case in which we were deterred from using this method—a case where meningitis followed its use.

In the case of a child between two and three years of age, who had nasal discharge, a mixed infection, staphylococcus unilateral with diphtheria bacilli constant, there were repeated cultures which were always positive. The house physician looked into the nose and found a shoe button there which was keeping up the local infection, and upon removing the shoe button it cleared up immediately.

We have had other cases where the culture removed remained positive in chronic rhinitis, which may possibly have been some ethmoid involvement. There are a number of points which might be brought out that are most interesting. One condition which we ought to consider is the occurrence of the postdiphtheritic paralyses. I do not know whether any work has been done in these cases or not. There was a question in my mind as to whether or not the constant harboring by the tonsil of toxins thrown out continually did not produce these paralyses. It would be interesting to investigate peripheral neuritis. I would like to do it if I get the cases, for I think it would be an interesting fact to determine.

We have made it a practice to give medication a thorough trial. We took the Shick skin reaction, operating only in six cases in which the reaction was negative. We have not used anything after operation. We have taken the nasal cultures and other cultures afterwards, so that our results would not be influenced by any application made to the throat. It is common in large hospitals for cultures to be taken after an application is made to the throat, and in the event of securing three negative results to let the patient go out. We determined to eliminate that factor entirely. The results after operation have not differed in any respect from those seen ordinarily. I reported several months ago six cases in which we had removed the tonsils and adenoids, where the results were startling. The next day after operation, without any application at all, the cases cleared. We took cultures for five days afterwards, and each time they were negative, except in one case, in which the nasal cultures remained positive.

The whole question is very important, and a simple reference to the literature will show a gradual wave in the curve of the amount of work contributed to the study of carriers in recent years.

Dr. Robert Clyde Lynch, New Orleans: My experience in this direction covers twelve or fourteen cases, and there are a few points I would like to bring to your notice. First of all, there are twenty or more varieties of pseudopathic bacilli in which the microscopist appears almost unable to absolutely and faultlessly tell the difference between the true organism and the pseudo variety. I think this is important, for frequently we might be dealing with a pseudo variety.

The second point is in relation to a little matter of technic in the administration of antitoxin in patients who have been previously sensitized by the administration of horse serum. The patient is given a small dose just above the ankle, and there is a constrictor ready for application above the knee; three or four drops are injected just under the skin above the ankle, and the constrictor ready. Sufficient time is allowed for the development of the anaphylactic symptoms, and if they do not develop in one-half to three-quarters of an hour, the injection can be made. If they should develop, the constrictor is applied.

I would like to report a case of a medical student, who had rather a virulent attack of diphtheria, which yielded to twenty thousand units of antitoxin. The patient gradually got well and finally had two negative cultures, with a normal temperature four days, and was ready for discharge. Suddenly there was a rise of temperature to 99°, and the appearance on the surface of the tonsil of acute follicular tonsillitis, which gave a culture of probable diphtheria. This was fourteen days after the first injection of antitoxin. We proceeded again to give him 120,000 units, and he was discharged. Six weeks later he came walking in on crutches, just able to get about, with paralysis of the soft palate and extensor muscles of the extremities, and suffering from constipation, which he had never had before. This man's cultures were taken and shown to be positive from the tonsils. He was treated locally without any result. Tonsillectomy was done, and in two weeks after the tonsillectomy the paralysis entirely disappeared without further medication.

Another case in which tonsillectomy was performed for carrier yielded nice results as far as the operation went. The cultures remained positive—nasal as well as throat cultures. We have taken cultures of both nostrils separately, and in this case we determined the discharge was coming from the antrum. We made a large opening into the anterior meatus and irrigated the antrum with buttermilk. The use of buttermilk has also cleared up some of the diphtheria carriers.

Another striking feature was that in nine out of the fourteen cases in which we could determine the form of organism coming from one or both tonsils, when the tonsils were removed we could not determine any organism in the tonsillar material. Yet all the cases cleared up immediately after tonsillectomy, and in none of these cases was the reformation of membrane in any way different from the usual case of tonsillectomy.

Dr. Robert C. Myles, New York City: This is a subject which interests me in a practical way, because of the fact that in many of the hospitals we frequently find children who have chronic nasal trouble with bacilli, without any other evidence of disease. The subject at hand would then be, what relation would a child having diphtheria carriers, but without any other evidence of diphtheria, bear to the other children in the home? I have had some very difficult propositions to contend with at times—sometimes the bacilli were found, and sometimes not, with the Board of Health inquiring, and the child is absolutely well and a perfect specimen of health at the time. I think this is a matter of important consideration. Of course, it is a purely local question, and our education of the Board of Health is one of the most important things in the matter.

Dr. Thomas Hubbard, Toledo (closing the discussion): All the study of the primary cases of carriers, without ever having clinical evidence of the disease, is largely institutional work, and to some extent also secondary cases. This latter group, however, have some diphtheria and are still diphtheria carriers. It is going to be put up to laryngologists to help them out in this matter, and by another year I hope to have records of many more cases and to arrive at definite conclusions.

Epithelioma of Posterior Pharyngeal Wall Cured With the Electrocautery.

By Dunbar Roy, M. D.,

Atlanta.

Female, aged twenty-seven years, first seen July 29, 1913. Previous and family history negative. Present history: For last three months had suffered with a soreness and throbbing in her throat. Had been treated continuously without result. Examination showed a rounded ulcer on the posterior pharyngeal wall at center, one-half of which was hidden by the soft palate; dirty grayish in appearance, with edges sharply defined; about one-half inch in diameter, and extending as deep as the superficial aponeurosis. A piece excised showed it to be an epithelioma.

Removal by means of the electrocautery point, well outside of its edges. No reaction and no discomfort followed. Healing perfect under one application. After three years there are no signs of a return.

Unfortunately, clinical observers are too prone to classify all malignant growths of the throat under the general term cancer, without distinguishing between the different forms of carcinoma and sarcoma. This statement is made because the writer has found it almost impossible to correlate all cases recorded, in that many of them were reported in the most unexpected places, and not under the headings where one would expect to find them. As Morell McKenzie and others have pointed out, the disease is often so extensive when first examined that it is impossible to tell its point of origin.

Textbooks are very vague in the discussion of this subject. It has now been three years since the case here reported has been healed, and there has been absolutely no signs of a recurrence. The results obtained in one case, especially of the cancerous type, certainly do not justify any positive deduction, but the writer believes that the thorough and judicious use of the electrocautery offers the best chance for a good result.

DISCUSSION.

Dr. J. Solis Cohen, Philadelphia: I have been very glad to hear the paper by Dr. Roy, because it exemplifies the opinion that epithelioma, taken early, before there is any glandular involvement, can be cured. The method which he adopted is an excellent one. In the first place, he eliminated the growth, and a certain portion of healthy tissue around it, with the cautery. The heat of the cautery extended and cooked some of the area around that growth, and entirely getting rid of the growth, he of course had a normal return of tissue. He got rid of the entire growth except as far as the scar tissue was concerned.

Some time previous to the twenty years' investigation which he spoke of, I very well remember a case in the clinic of the Jefferson College Hospital. It was a case in which there was a spontaneous cure of a growth similar to the one mentioned, about the size of the end of the thumb. A portion was removed with forceps for examination, and in the interval waiting for the report the patient was allowed to go home. I powdered the area and let him go; he was a carpenter, sixty years of age. The man was instructed to return at the next clinic, and when he did so he looked better, and nothing else was done. The specimen was reported as epithelioma. I do not, however, attribute it to the powder, but have always looked upon it as a case (such rare cases have occasionally been reported) of spontaneous retrogression of a pharyngeal epithelioma.

DR. CORNELIUS G. COAKLEY, New York City: I think the case of Dr. Roy is exceedingly interesting. It presents one phase, however, to my mind, which is a little doubtful, and that is, that I understood the patient was examined only in frozen section. I think most pathologists will feel a little bit in doubt as to their examination of a frozen section.

The next point is the unusual absence of glandular involvement in this area. I have never seen a malignant process, even fairly early, on the posterior pharyngeal wall, that did not have the glands involved. It may have been some particular type of cancer—some peculiar form, different from the average type which we ordinarily see.

The next point is one which I think we ought to take up seriously, and that is the absence in the literature of records of cases of involvement of the nose, throat, accessory sinuses, pharynx and larynx. Within the last year I personally ran across at least one dozen cases of malignant disease of rather unusual type—ethmoidal, maxillary, etc. I had one case recently involving the sphenoid and nasopharynx—whether primary or secondary, I could not tell. Those cases are unrecorded in the literature, and I have no doubt that those of us who are working in large clinics have numbers of these cases. I think we should report all cases of malignancy seen during the year and have them tabulated in some form for reference. It is really surprising to note the relative frequency of these cases as compared with the actual records.

DR. D. BRYSON DELAVAN, New York City: There is nothing so uncertain about the prognosis in a case of socalled epithelioma. I say "socalled," because we cannot get further information histologically than the microscope is able to give us. When the microscopic examination is made and the findings reported, there our information ends. There are in the recollection of most of us isolated cases which differ entirely in their result from the average. For example, the solitary case upon which Dr. Elsberg operated at different times for a dozen years, and upon which Dr. Cohen operated twenty years after its inception, in which the patient lived fifty years after the first operation. That is a long while for a patient to survive epithelioma.

In contrast to that which represents a type now and then spoken of and recorded amongst thousands of others, I had a case of epithelioma of the epiglottis which was discovered so early that the growth was only about half the size of a split pea, one-quarter of an inch in diameter, on the extreme margin of the epiglottis. In that case one-third of the epiglottis was thoroughly removed, as far down as possible, and the disease apparently completely extirpated. After four or five years there was another mass there—a perfectly normal healthy condition—and then the patient redeveloped the disease, from which he died after a comparatively short course.

Now, that case is instructive because it shows that if we operate, as we suppose, far and wide out of the lesion itself, nevertheless structures in that neighborhood may have already become involved after all, and with the lapse of time the involvement will appear and the disease go on, in spite of all treatment, to the destruction of the patient.

We know about this subject and the number of such cases that have been studied all over the world, and reference to the poverty and misleading nature of the statistics is very timely. I called your attention yesterday to the work of the Society for the Prevention of Cancer. I wish now to bring that before you again, and to urge as forcefully as I can the desirability of carrying out the suggestion of the last speaker. As chairman of that association, I can say that the association stands ready to give all the aid it can to any institution which may interest itself in the matter of statistics. Apparently, this is now the thing most needed. I hope this society will bear that in mind. Each man here is a central influence in the place where he lives, as well as in the vicinity of that place, and I hope each man here will do the best he can to help forward this work for the study of cancer.

Dr. Dunbar Roy, Atlanta (closing the discussion): In reply to Dr. Coakley, there was a frozen section made for diagnosis, and later on this was confirmed. The case was operated upon, however, under the diagnosis of the frozen section.

I have nothing further to add, except the fact that anyone who has had any of these cases and tried to look up the literature, will know what an awful burden it is. It is almost impossible to get anything whatever. With the majority of writers "cancer" seems to be a general term, used to cover everything; one gets into a labyrinth of cases from which nothing can be made out. The words of Dr. Delavan are very timely, indeed. We certainly ought to be a little more scientific and get every case recorded as far as possible, as from its point of origin.

Extensive Cholesteatoma Following the Luc-Caldwell and Killian Operations, Simulating Sarcoma. Case Report.

By Virginius Dabney, M. D.,

Washington, D. C.

Man, forty-two years of age, gave no subjective symptoms of his grave condition other than nasal stoppage. Exophthalmos marked, deviation of septum complete, polyps in middle strait. Acute exacerbation three days later. Luc-Caldwell operation and extensive exenteration of ethmoid bone, with subsidence of symptoms. Five days after this, symptoms returned, and Killian operation done, with perfect functional and cosmetic results. Eleven days later abscess in cheek formed and was evacuated by incision below and parallel to the lower eyelid. Great distension of cheek, frequent spontaneous hemorrhages, convincing radiographs and wooden-like hardness of mass in cheek suggested sarcoma. Two months later, operation revealed immense collection of true cholesteatoma; odor overpowering; all bone above, below and on each side of mass eroded and totally destroyed, including floor and inner wall of orbit, two-thirds of malar bone and all of outer wall of antrum. Present condition of patient, marked asthenia; death only matter of short time. Syphilis, tuberculosis, malignancy, all excluded by proper methods.

DISCUSSION.

Dr. Oscar A. McKimmie, Washington: I had the privilege of seeing this case a number of times with Dr. Dabney before operation, and of being present and helping him with the operation which he has detailed. He has given a very complete report of the condition as it existed, and there is very little I can add about the case except to explain that the condition might very readily have given rise to a misleading interpretation of the X-ray plate. The superior process of the bone was the thickest bone I have ever seen in living subject or in dead. One portion of it was at least one-half inch thick, solid and ivory-like in character, and these cells in the ethmoid which Dr. Dabney has referred to, I have never seen in a living or dead subject. They were very hard and extremely thick, and this explains very readily why at the operation it was impossible to get away all the cells. At the secondary operation it was shown to be a very dense bone, either an anomaly of development, or the result of chronic inflammation.

The question of the origin of the cholesteatoma is an interesting one. There is no question as to the character of the mass, because all its clinical appearances and the laboratory findings proved it to be cholesteatoma. The mass taken out at the secondary operation, I should say was as much as one could hold in the hand. The swelling from the malar bone to the bridge of the nose was a perfect straight line, and the odor was something that I have never experienced in an operating room before. My own impression is that this secondary infection, of whatever type it was, which was associated with this sinus, was probably the starting point of the cholesteatoma formation.

Dr. Joseph H. Bryan, Washington: I have seen one or two instances where there was ivory hardness of a white, previously soft mass, where we found suppurative conditions of long standing. It probably was an osteitis which eventually produced this cholesteatoma mass.

Dr. Virginius Dabney, Washington (closing the discussion): I did not go into details about how I arrived at the conclusion, except that it was examined under the microscope by a pathologist. Cholesterin crystals were found, and this would confirm the diagnosis. I think Dr. McKimmie's theory is a very tenable one. The hole was right here, and we never made it heal; nothing would make it heal; it is open now.

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XVII.

A SURGICAL CONSIDERATION OF THE UPPER PARANASAL CELLS.*

BY GREENFIELD SLUDER, M. D.,

ST. LOUIS.

I propose a surgical procedure for the upper paranasal cells which cuts downward and forward. All other procedures, so far as I know, cut more or less upward and backward.

Anatomically the paranasal cells are designated according to which meatus of the nose they enter. This is more or less an anterior and posterior subdivision, and for the purposes of diagnosis this must remain unchanged. For surgical purposes, however, it seems to me that they may advantageously be thought of as upper and lower, the latter being the antrum of Highmore, the former being the remaining cells. This subdivision establishes a horizontal dividing line on the lateral wall at about its middle. Successful surgery of the antrum must leave a drain at the level of the nasal floor. The lowermost part of the upper cells is along the line separating them from the antrum—i. e., the horizontal midline. Successful surgery of the upper sinuses, however, is not achieved by a drain along this line, for the reason that there are usually cells which are limited to a part far above the midline. Successful surgery of the upper sinuses must lay open for drain the upper cells in their entirety, beginning at or very close to the cribriform plate of the ethmoid—the roof of the nose—opening at the same time the lowermost of these cells—that is, removing the lateral wall of the nose with the middle and upper turbinates above the horizontal midline.

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*Read before the meeting of the American Laryngological Association, May, 1916.

This procedure, it will be readily conceived, may be conservative or may be made radical. The former consists of the removal of the turbinate and the very innermost layer of the capsule of the ethmoid. The radical operation consists in removing the capsule "part and parcel," including the orbital plate, when desirable for drainage of the orbit into the nose, at any or all its points, from its anterior limit to its apex.

To this end particularly was directed the understanding mind of H. P. Mosher when he proposed the "surgery of the ethmoidal labyrinth." This procedure must of necessity be a radical one, although he does not recommend it for the extreme of radical—opening the orbit.

Also to this end was directed the fearless effort of W. L. Ballenger when he proposed the exenteration of the ethmoid.

It seems to me that the procedure I herewith submit has some advantages that neither of the others offer. First, Mosher's operation cannot (as I understand it) be made conservative. By this I mean the simple freeing of the outlets of the cells, leaving their walls otherwise intact. And the more completely it is executed the more risk it must needs involve. In the hands of anyone less familiar with it than Dr. Mosher, it must become more serious the closer he approaches the uppermost nasal limits.

Second, Ballenger's ethmoidal exenteration requires as its primary requisite, soft bone—that is, bone that has been softened by disease—and, furthermore, it requires a lateral wall that is not a plane. Should the lower half of the lateral wall extend outward in a way more or less under the ethmoidal capsule, the special knife he so ingeniously devised may be readily engaged from behind and cut more or less into the capsule in its forward and upward pull. I cannot, however, conceive of it opening ethmoidal cells anterior to the uncinate process, because the direction of the blade changes in this part of the nose so as to defeat this; and the uncinate process is usually, if not always, so hard that it will cast the knife back into the free space of the nasal fossa. It will turn it off as a chisel may be turned off by a nail in a piece of wood. Should the lateral wall be approximately a plane with the turbinate set on it, the knife will remove the posterior four-fifths of the turbinate only, unless the wall be exceedingly soft, when it might bite into the capsule. But for a nostril with many polyps and swollen capsule made of cells filled with pus, Ballenger's exenteration has many advantages—e. g., the instrument need be introduced into the nose only once, which helps much with terrified patients; and it may be done quickly. For these reasons I prize it and use it especially where the case is ethmoidal without frontal or sphenoidal elements.

The procedure I describe has been satisfactory in my hands for thirteen years. It may be limited for the frontal sinus, providing a very high cut of the middle turbinate, which I have frequently mentioned in various texts as a "cribriform turbinectomy." The cut is actually two or two and one-half millimeters from the cribriform plate, and may be extended to the most anterior limit of the infundibulum. Or this very high cut may be carried backward to include the capsule of the ethmoid, under which conditions not only is the middle turbinate removed, but the uppermost line, and usually all the other ethmoidal cells are opened wide into the nasal cavity. And when desirable the entire anterior wall of the body of the sphenoid, from its uppermost limits, with all its postethmoidal association and much of its floor, may be removed. In my judgment and experience it is the technic that most often may be trusted to open all of the cells, regardless of unusual or anomalous positions. I know, however, that cells may exist that not only cannot be opened from the nose, but cannot be located by any means at present at our command. They can only be recognized in the cadaver, and then only by complete dissection. Everyone who has observed a reasonable number of specimens must have seen such cells and have been struck by the futility that would attend any surgical effort to reach them; and everyone of reasonably wide experience has met cases where all efforts had failed to find and treat such cells. But there are many other cells that are placed in positions more or less unusual that will be opened by a technic which has for its primary plan an incision which will skirt the cribriform plate and remove the middle turbinate at its most anterior as well as its uppermost limits, and extend into the sphenoid body at its uppermost part, regardless of the natural opening, and then be extended downward until it has cut through its floor or found it to be impenetrable. I have here avoided the designation of sphenoid sinus, because such a term does not always comprehend the full body of the sphenoid. It (the body) may be subdivided and shared by a postethmoidal cell. And it is this variation which I believe to be the most pernicious from the clinical side (for headache and optic nerve lesions) as well as anatomically far more frequent than is at present recognized clinically. This variation will (it seems to me) be satisfactorily dealt with by this proposed technic—practically every time it is met. Also prefrontal ethmoidal cells will be opened into the nose as a rule, if not uniformly.

The Operation.—In 1907 I published an elementary text in which I described a surgical method which was at that time novel. It consisted in approaching the turbinate from above on its inner side. Prior to that all surgical approach was from below laterally upward, removing it by scissors or snare or a combination of these or such working instruments. I described a knife consisting of a handle, a shaft and a cutting end turned at a right angle to the shaft and sharpened so as to cut on the inside of the right angle, i. e., on the pull. It was also sharpened on a face parallel to the shaft, and at the same time gave it far more strength than a hook must have were it sharpened on its concavity. I selected this shape in preference to a hook for these reasons, and the fact that this rectangular form is much stronger than a hook; and that great strength is necessary for the tasks to which this knife is put. The knife, although possessing great strength, is so small that it may readily enter spaces which larger instruments could not. I believe it to be the smallest nasal instrument possessing great strength.

Surgical Procedure.—A glance at the lateral wall shows that the higher and the more forward the infundibulum is opened, the wider and freer will be the inlet to the frontal sinus usually. At the same time prefrontal ethmoidal cells will usually be opened by such an incision. For these reasons it has been advantageous in my hands to introduce the knife sagittally under the anterior third of the middle turbinate as far back as the uncinate process, as high as the cribriform plate, with its cutting edges facing forward. It is then rotated somewhat inward and drawn forward and downward. (Fig 1.) Considerable forward pressure is kept on it while it is being drawn downward. In this way the foremost and uppermost nasal wall of the capsule of the ethmoid (with the attachment of the middle turbinate) is cut open and the prefrontal cells of the ethmoid are thereby thrown open, as the nasal wall of these cells is usually constituted by this part of capsule with the attachment of the turbinate. This must be considered the conserative technic. By these means, however, is put an outlet to the usual frontal sinus that comprehends the dimensions of the usual infundibulum—i. e., one centimeter anteroposteriorly and one-half centimeter laterally. Should this size be reduced to such a degree as to require some procedure more radical, the knife is then engaged in the hiatus semilunaris, with its cutting edges facing forward or downward—usually in the uppermost limit. By a down pull the uncinate process will be removed and the knife may finally be engaged in the floor of the anterior part of the frontal sinus. (Fig. 12.) This is usually so thick and hard that it is with difficulty or impossibility handled by this knife. A large enough opening, however, is usually secured to admit a Good rasp or a burr. The burr is then introduced into the sinus and put in motion with a forward and downward pull. In this way I have slightly modified Ingals' ideal frontal sinus technic. This stroke of the right angle knife it will be seen is safe, as it cuts away from danger; or the knife in position, cutting edges facing upward and forward, is likewise safe with a downward pull, as it also cuts away from danger. It will cut into the buttress of the lower anterior part of the floor of the sinus.

For the ethmoidal district the knife is reintroduced along the cribriform plate—its smooth elbow with a gentle sense of touch, recognizing the smooth uppermost limit of the nose—to a distance of one centimeter. In this position its two cutting edges face downward. It is now rotated till its cutting edges point outward and slightly upward. The upward turn is added to the outward for the reason that the outer (horizontal)position permits of a straight line pull downward and forward, and this direction is not easily changed. This is at once away from the cribriform plate. The slight upward turn keeps the line of incision much more nearly the horizontal, despite the fact that the pull must needs be downward and forward or forward and downward. The safety of this cut is provided by two factors—i. e., the cells of the ethmoid in their extensions into the roof of the orbit rise at once above the cribriform plate; and the length of the cutting arm of the knife, which should be two millimeters, is too short to cut through thickness of the nasal orbital thickness of the ethmoid capsule under any circumstances. This incision is the first of two or three similar ones. (Figs. 2 and 3.) I prefer to divide it into two or three because I can keep my bearing upon the cribriform plate more satisfactory by these shorter cuts than in one longer cut.

Now the operative procedure is to be directed by the judgment of the surgeon. In my judgment the only turbinectomy, according to my experience, should be a cribriform or infundibular turbinectomy—i. e., one that opens the infundibulum to its widest possibilities, for the reason that a lower cut leaves a stump that is physiologically worthless and surgically difficult of later manipulation. This is not in reality a stump, although in former years I considered it such. It is that part of the ethmoidal capsule which makes the inner portion of the final entrance of the frontal sinus into the nose—the anterior part being the uncinate process, the posterior part being the bulla ethmoidalis, the external and internal parts being ethmoid capsule. The removal of this inner part as high as possible usually gives an inlet to the frontal sinus six millimeters anteroposteriorly by three millimeters laterally. Some frontals enter the nose by a devious* channel and cannot be considered as usual. If this upper internal part of the frontal inlet be not removed at the primary operation, it seems to me to be unfortunate, as a secondary operation will almost surely wound the epithelial surfaces of the frontal inlet and then be followed by connective tissue development which will block the inlet. The primary high cut is usually accomplished without that result, leaving the hiatus semilunaris and infundibulum free of scar tissue. The secondary operation is usually of necessity done with more trauma to the hiatus, regardless of whatever technic be employed. For this reason I emphasize the advisability of doing the foremost cribriform cut primarily if the anterior half of the turbinate is touched at all surgically, regardless of whatever else is to be done surgically.

The cutting edges of the knife are now turned downward, or downward and slightly outward (Fig. 4), and the incision is placed superficial (internally), or may be placed as deep (externally) in the capsule of the ethmoid as the judgment of the surgeon directs. For the simplest subacute suppuration cases the superficial incision suffices. For most radical—e. g., an orbital phlegmon back of the transverse meridian of the eye—the incision goes through the capsule into the orbit in its posterior half. This is readily and quickly done. The detached mass is then by means of the same knife, which may be turned somewhat transverse, pushed downward into the nose, where a snare may be put around it and cut it from its attachments. (Fig. 5.) The procedure may be varied by a superficial removal of the turbinate and then the excision of the capsule to any extent desired. In my practice I have had twenty orbital phlegmons, produced by nasal sinus ruptures, twelve of which I opened into the nose in this way back of the transverse meridian. In my experience this has far surpassed the results of external orbital drainage, inasmuch as it drains by the same route that infected the orbit.

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*Hyman and Ritter.

Should the sphenoidal and postethmoidal cells also need the surgeon's care, I then (after the snare amputation of the middle turbinate) pass the angular knife very gently backward along the cribriform plate, with its cutting edges downward. In this way its smooth round elbow glides along the cribriform plate until it meets the anterior face of the body of the sphenoid. This it will be seen is the uppermost point of the face, and is independent of the natural opening of the sphenoid sinus, which is usually well below this point.* Gentle pressure is now made on the face of the sphenoid and the knife inclined slightly toward the septum. The first stroke perforates the usual sphenoidal face and cuts somewhat downward. The knife, cutting edges downward, is then introduced very gently into the opening so made and very gently extended as far back as the cell permits. It is rotated fifteen degrees inward, and downward with any degree of force that may be needed. This incision will extend from the junction of the cribriform plate with the body of the sphenoid downward a varying distance along the septum nasi, depending on the thickness and hardness of the bone. Should this incision not have reached the floor of the sphenoid, another similar one usually does. (Fig. 6.) The knife, edges down, is again passed along the cribriform plate and very carefully introduced back into the uppermost limit of the opening already made. It is then rotated thirty degrees outward and brought out forward and downward. (Fig. 7.) This incision includes a considerable part of the wall separating the postethmoidal from the sphenoidal cell, and it has the advantage of beginning at the uppermost limit of the sphenoidal face. It has a distinct advantage of all methods which begin their procedure in or from the natural opening of the sphenoid, for the reason that often a postethmoidal cell occupies a part of the body of the sphenoid and is placed above the sphenoidal cell. Fig. 13 shows such a cell which would be opened by this technic and would probably not be opened by any other technic now known. Such cells are exceedingly hard to deal with surgically and sometimes equally hard or impossible to find. Fig. 14B shows such a cell placed all above the line of the cribriform plate. This cell would not be opened by this technic, and I do not believe it could be opened by any other technic. Moreover, I do not believe it could be recognized in life by the present means of investigation. Some such cells, however, have a lateral and downward extension which is opened by the second stroke of the knife, which is again introduced along the cribriform plate and inserted into cavity in the body of the sphenoid, turned thirty degrees outward and drawn forward and downward.

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*Loeb.

This second cut usually takes the postethmoidal nasal wall, as well as most of the lateral part of the sphenoidal face, and often the mass may be removed in one piece. For this purpose I use the Knight forceps (Figs. 9 and 10), made, however, for this particular purpose, being two centimeters longer than the original model. These forceps are then introduced into the cavity of the sphenoid (Fig. 8), and so the floor of the sphenoid is removed in part. Everyone knows, however, that the floor often is so thick and hard that it cannot be removed by forceps. A drill which will do such service may be advantageous.

In the performance of the postethmoidal sphenoidal portion of the technic I prefer a knife smaller and slightly different from that used in the anterior portion, for the reasons that it is thereby rendered much stronger; it cannot be broken in any of the service it may be put into, and secondly, a knife 1.5 millimeters inside cutting edge cannot inadvertently cut into the orbit in this service unless wielded by some one neglecting the proper performance of the operation and totally ignorant of the anatomy here; and third, I feel that the angle of the sphenoid knife should not have turned the full ninety degrees from shaft, because such a knife could be only with difficulty engaged in the face of the sphenoid; an angle sixty-eight degrees off the shaft, which will make the cutting edge on the inside of the angle one hundred and twelve degrees, will bite readily into the face; and fourth, a small knife 2.5 millimeters in its entire cross measurement permits a much closer approach to the tip of the sphenoid face. One 6 millimeters could not get so close by 3.5 millimeters. In this service a hook sharpened on its concavity would be much less advantageous, because, if bent to a right angle, it would not bite into the face, and if in sixty-eight to one hundred and twelve degrees shape it would not execute the work so well and its point would break easily. It would then take the shape of the knife now proposed.

I feel that special emphasis should be laid upon the necessity of the surgeon knowing what are the correct models of these knives. For this reason I submit accurate drawings. Too large a knife has the disadvantage (1) of taking up space unnecessarily and of (2) biting more bone than it can cut, and (3) of possibly cutting inadvertently into the orbit, and (4) of being much more apt to have its angle cutting portion broke off in the operation. In this connection it must be emphasized that it is absolutely necessary that the knife be not tempered too hard. Should it be too hard it will always break with the work put on it in this operation. The instrument maker must specially understand this. It is far better to have it too soft, and though its cutting edges may not be as lasting, it will not break.

SUMMARY.

The intranasal surgery of the upper cells may be performed by this method in any part or the whole as conservatively or as radically as desired. The ability to place the incision two millimeters below the cribriform plate with safety in any part or in the whole length of its extent seems to me to be most advantageous, and not a small part of this is the power to extend this incision to the foremost limit of the infundibulum, thereby opening the inlet of the frontal to its widest natural possibilities. It is most desirable to preserve the natural inlet here, and this is done by a cribriform turbinectomy which leaves undisturbed the histologic epithelial covering of the normal inlet—i. e., the uncinate process, the bulla ethmoidalis, the hiatus semilunaris and infundibulum—regardless of the anatomic variations of the frontal inlet. Should these parts be wounded, as in a curettement, the resultant scar tissue blocks the inlet. The angle knife removes by cutting any desired tissue with the least possible trauma to the surroundings. In the sphenoidal district it opens uppermost and lowermost possible parts of the wall, which has the advantage sometimes of opening also a postethmoidal cell which may occupy part of the body of the sphenoid. (Such a cell is often the cause of the entire clinical picture). The angle knife is so small that it takes up the minimum of room, and so leaves the small field open to the best vision possible. Its execution is always in the direction away from the danger zone. I have so far not seen such satisfactory postethmoidal surgery by other methods. (This district seems to me the most dangerous of all, for on the outer upper aspect runs the optic nerve, and above is the optic chiasm and cranial cavity). Satisfactory execution here is necessary, particularly in eye lesions.

The entire performance may be accomplished within a short time. I have often finished the high frontal ethmoidal and sphenoidal combined operation in two minutes (including the postorbital opening on one occasion). A septum nasi deflected into the affected side may add troubles for the surgeon. I have in such cases where the middle turbinate was not visible from in front in any part used a bivalve speculum specially constructed for the purpose. Just as Killian elongated the blades of the primary bivalve for his needs in septum resection, I have elongated them still more and widened them for my needs here (Fig. 7). The speculum should be made of tempered steel blades, knife-like, thin, with handles long enough to give leverage to dislocate the entire septum into the opposite nostril. It should have a set screw, for the pressure required is much and the hand gets tired holding it. It should have blades nine millimeters wide, for narrower ones, such as are supplied with Killian's, often do not give sufficient view. They should be eighty-eight centimeters long, for shorter ones (Killian's) sometimes fail to reach the posterior field of operation.

The knives are also made right and left (Figure 16). Some of my associates prefer these. My own preference is for the single, straight, original model. I recommend an extra two and one-half centimeters long shaft for use with the long blade sphenoidal speculum. A strong, large handle ought always to be supplied for the knife.

A right-hand surgeon makes his turbinate cuts for the left side, cutting edges in the place of the handle. For the right side the cutting edges are turned one-quarter (ninety degrees) into right nostril. This makes them horizontal or transverse when the handle is on the middle line.

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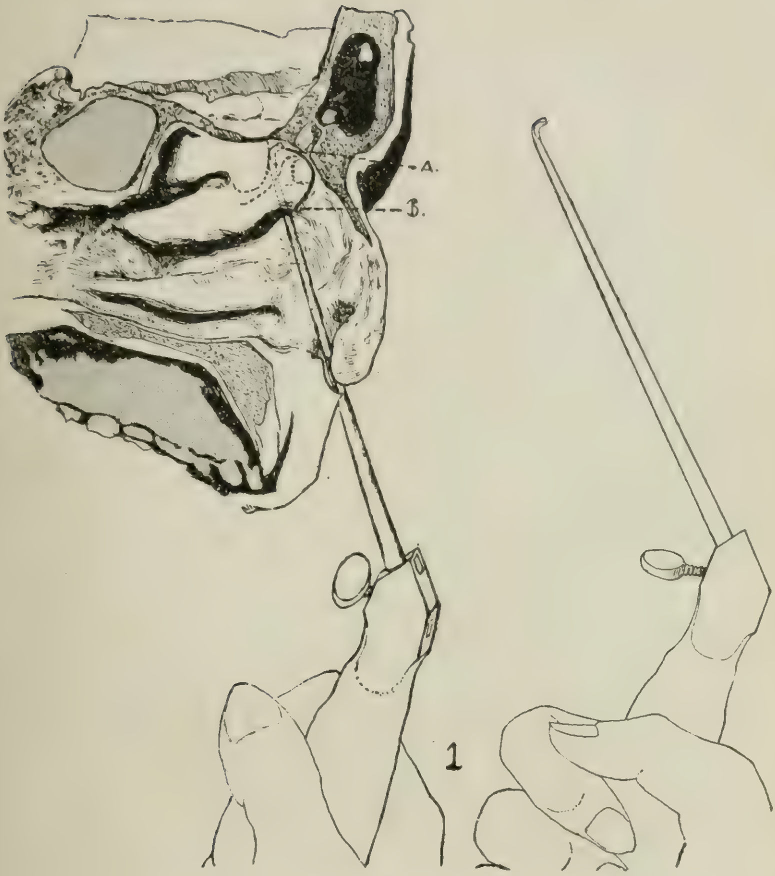

FIGURE 1.

On the right the angle knife is shown in the sagittal plane, ready for insertion under the middle turbinate. On the left it is shown in position for the forward and downward cut. It has been introduced into the infundibulum and rotated thirty degrees inwards— i. e., toward the septum nasi. The heavy line a-b shows diagrammatically the line of this first cut, which may be accomplished in one stroke or two, according to the surgeon's election. (I prefer two strokes usually.)

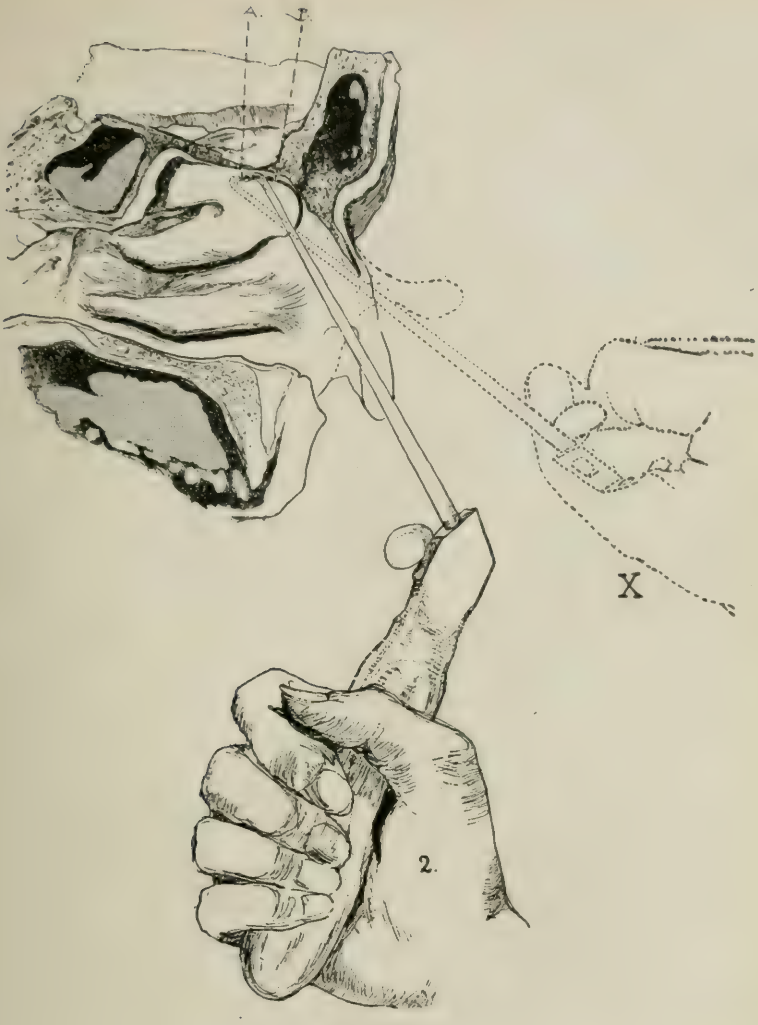

FIGURE 2.

Shows the angle knife introduced between the septum and middle turbinate. It has passed one centimeter back of the first cut along the cribriform plate. The dotted line X shows it to have been rotated outward until its cutting edges point thirty degrees above the horizontal. The tip of the nose has, at the asme time, been forcibly elevated. The heavy dotted line a-b shows the line of cut.

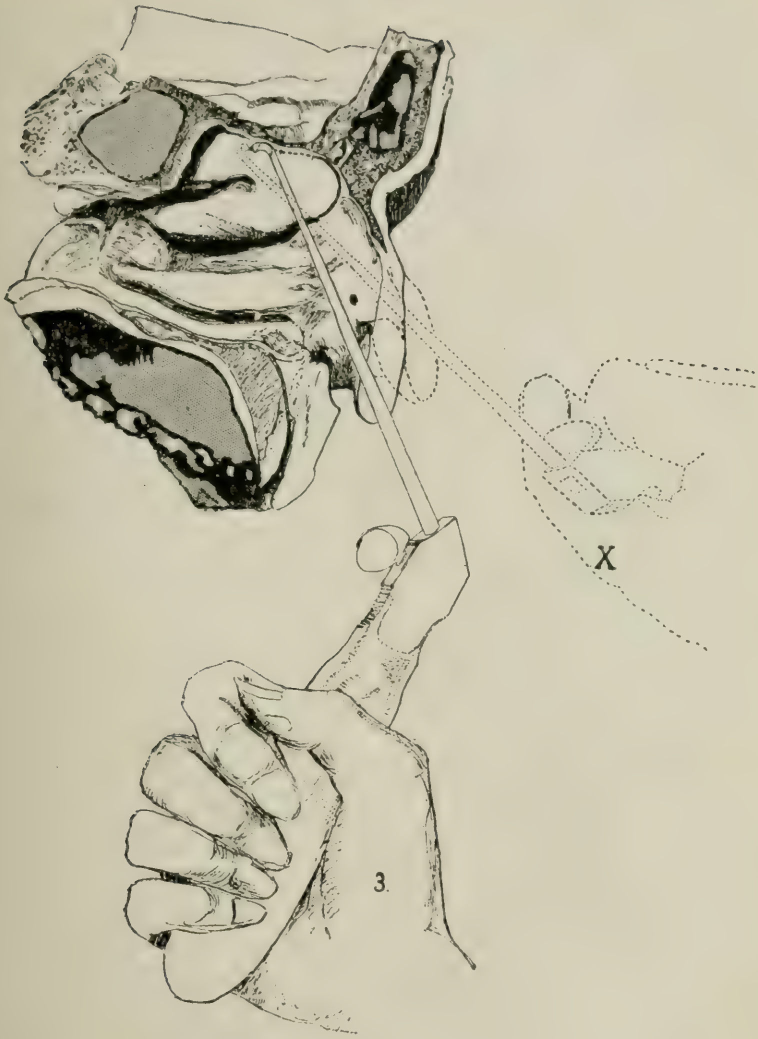

FIGURE 3.

Is the same as Figure 2, with the difference that the knife has been passed the second time along the cribriform plate to a distance one centimeter back of the first cut.

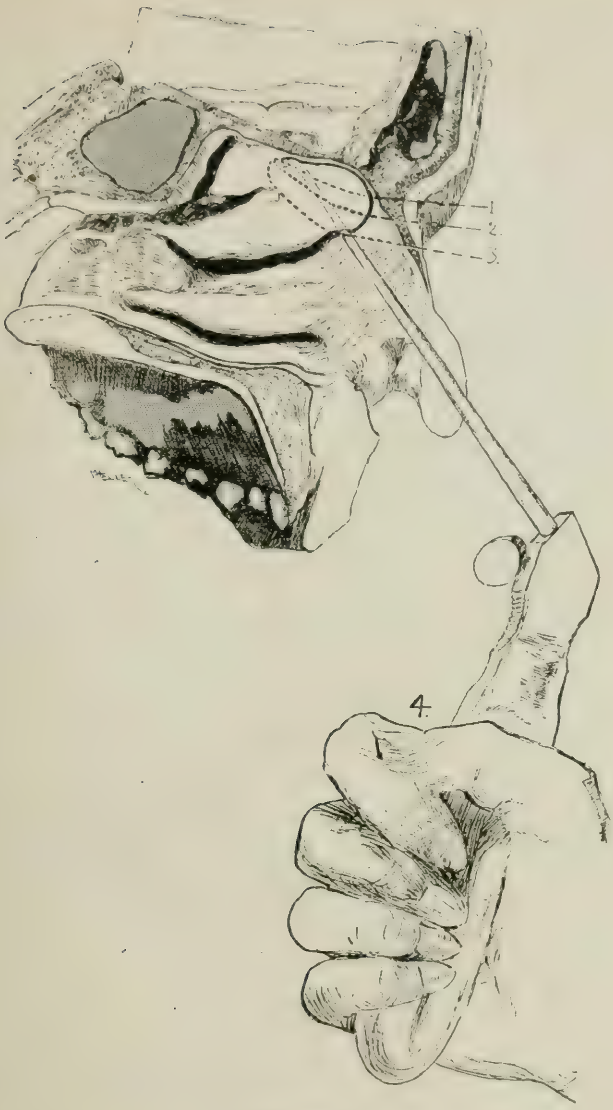

FIGURE 4.

Shows the knife introduced sagittally under (lateral to) the middle turbinate for the purpose of cutting into the capsule of the ethmoid. The dotted lines 1-2-3 show the successive cuts from above downward. These cuts may be readily made to enter the orbit by inclining the cutting edges thirty degrees outward. In the conservative employment of this technic, this employment of the knife may be omitted.

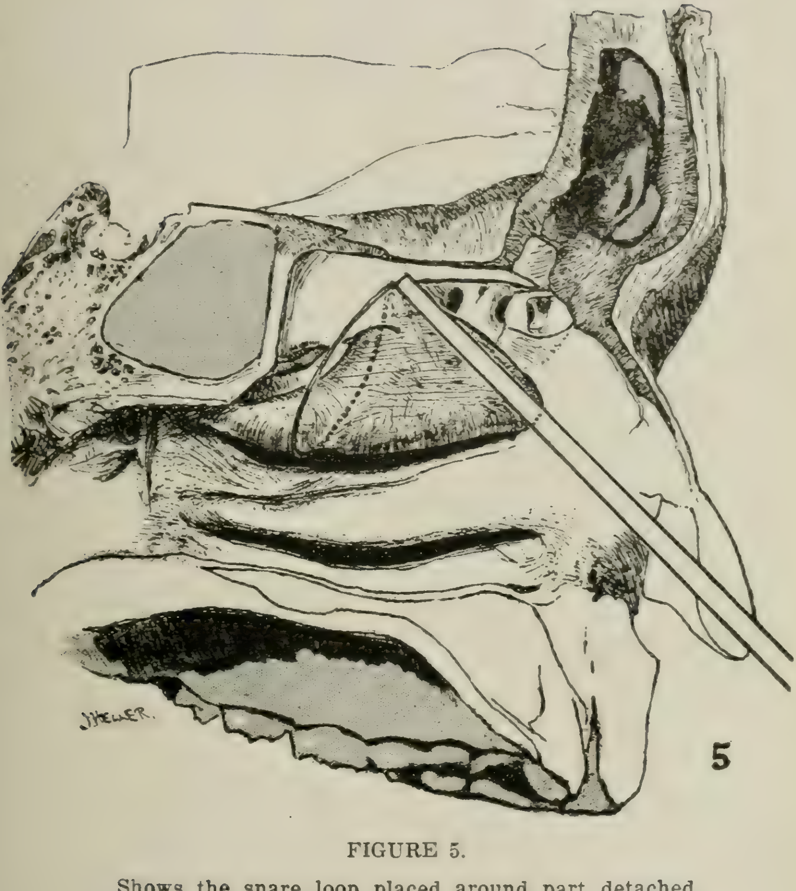

FIGURE 5.

Shows the snare loop placed around part detached.

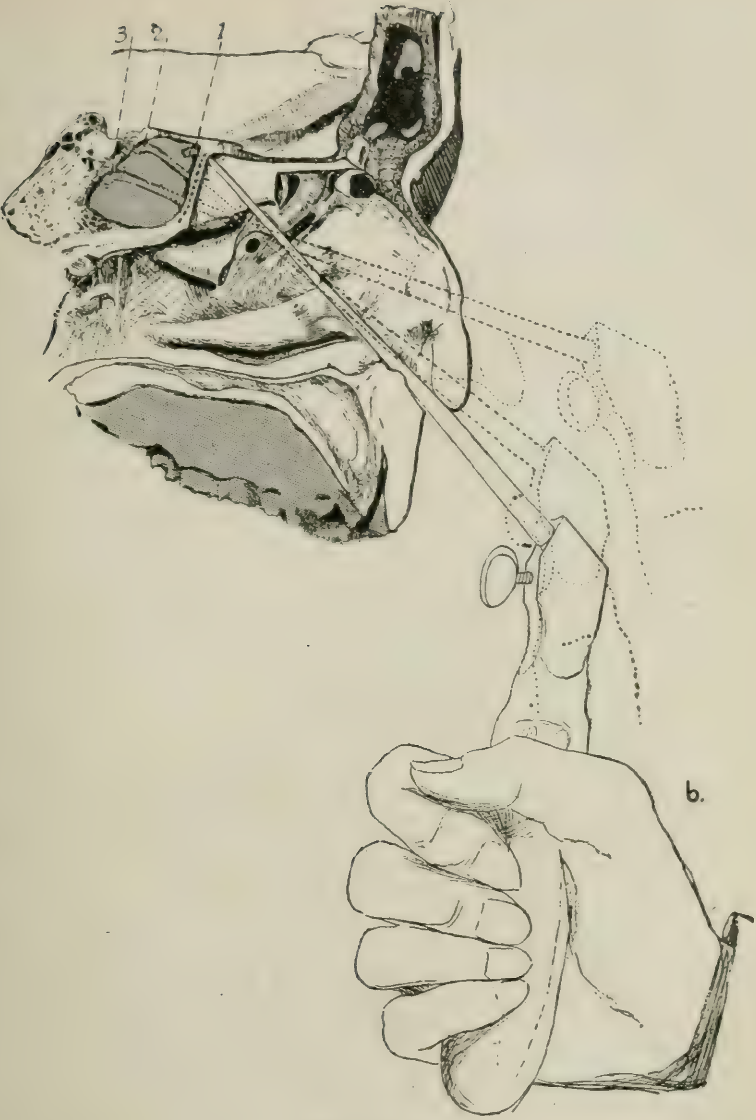

FIGURE 6.

Shows knife 1 passed backward along cribriform plate, penetrating anterior face of sphenoid at its top. The dotted lines 2 and 3 show knife passed back to posterior wall of cell and to have been rotated twenty degrees inward, cutting downward to hug the septum nasi.

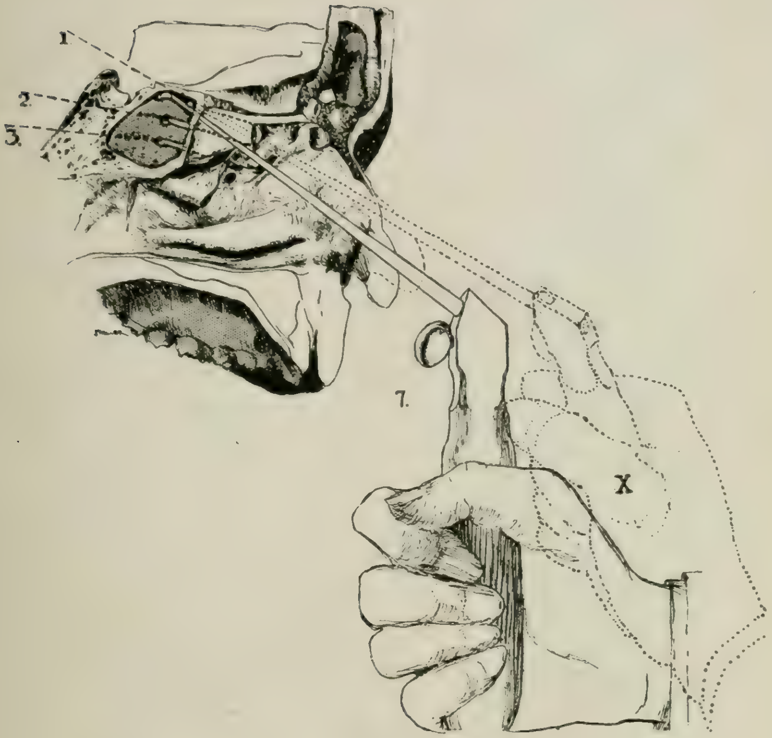

FIGURE 7.

Shows knife to have been reintroduced through uppermost part of sphenoidal cut, as shown in Figure 6, to posterior wall of cell. It is then rotated thirty degrees outward (X) and carefully drawn forward and (necessarily) downward. The introduction of the knife to the posterior wall of the cell of its uppermost part insures the opening of many of the postethmoid cells that are developed into the sphenoid body. I believe this to be very important and one of the advantages to be secured by this technic.

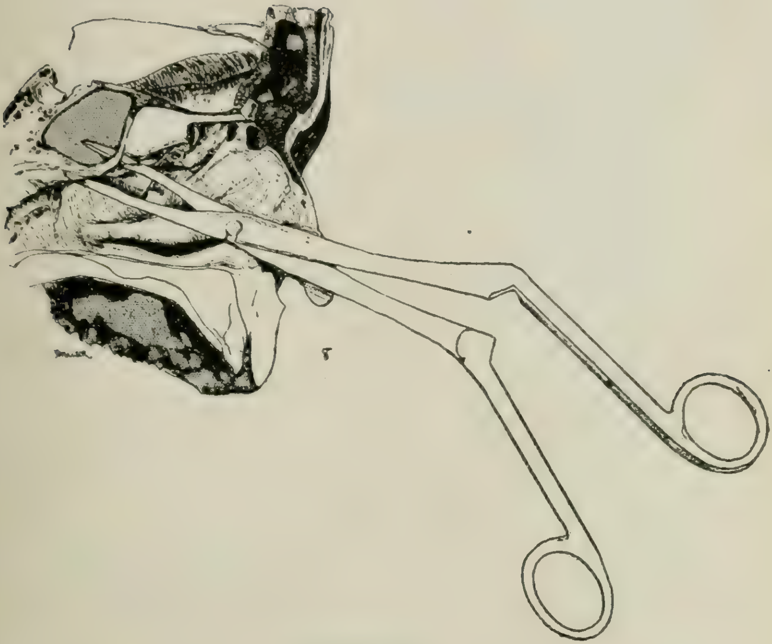

FIGURE 8.

Shows the Knight forceps (made especially for this purpose three centimeters longer than the original model, because this added length is often required) introduced into the opening in the anterior face of the sphenoid body ready to bite out the anterior half of the floor of the body. This is oftentimes easily done, but at other times the floor is too thick to be removed this way. It is removed by a rotatory movement.

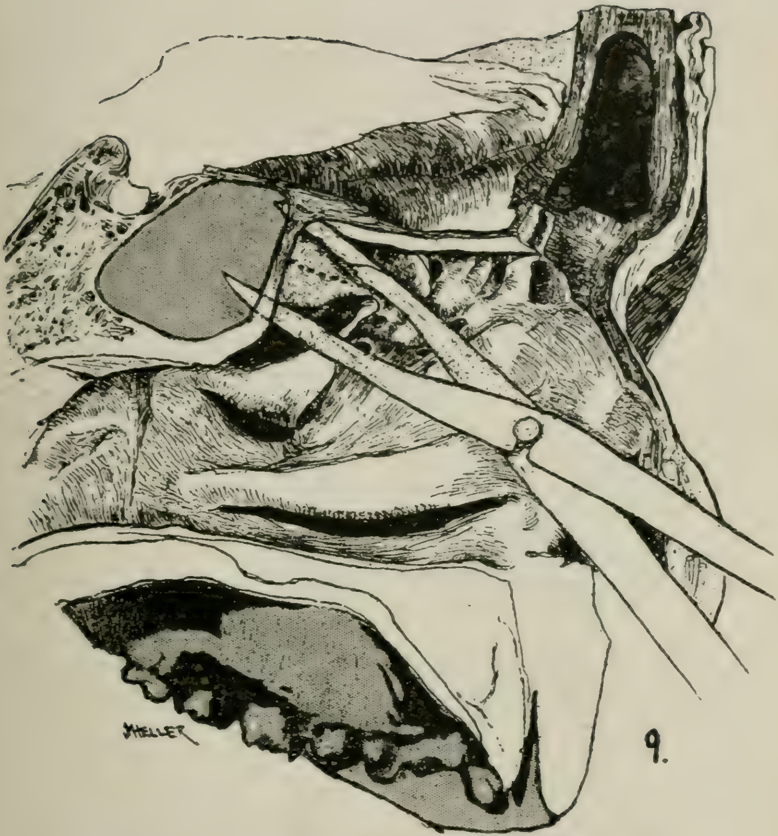

FIGURE 9.

Shows the Knight forceps in position to bite out the postethmoidal wall which has been cut loose, as shown in Figure 7.

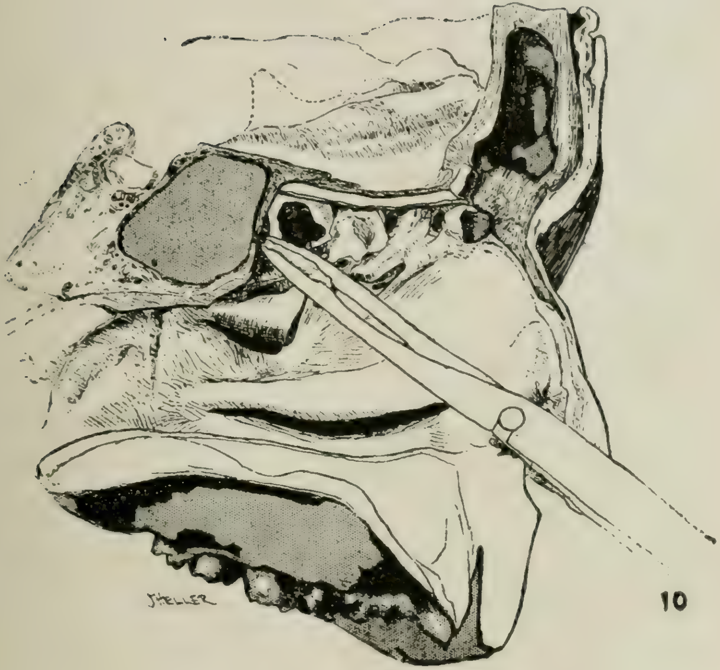

FIGURE 10.

Shows postethmoidal wall in grasp of forceps. It is finally detached by a rotatory movement

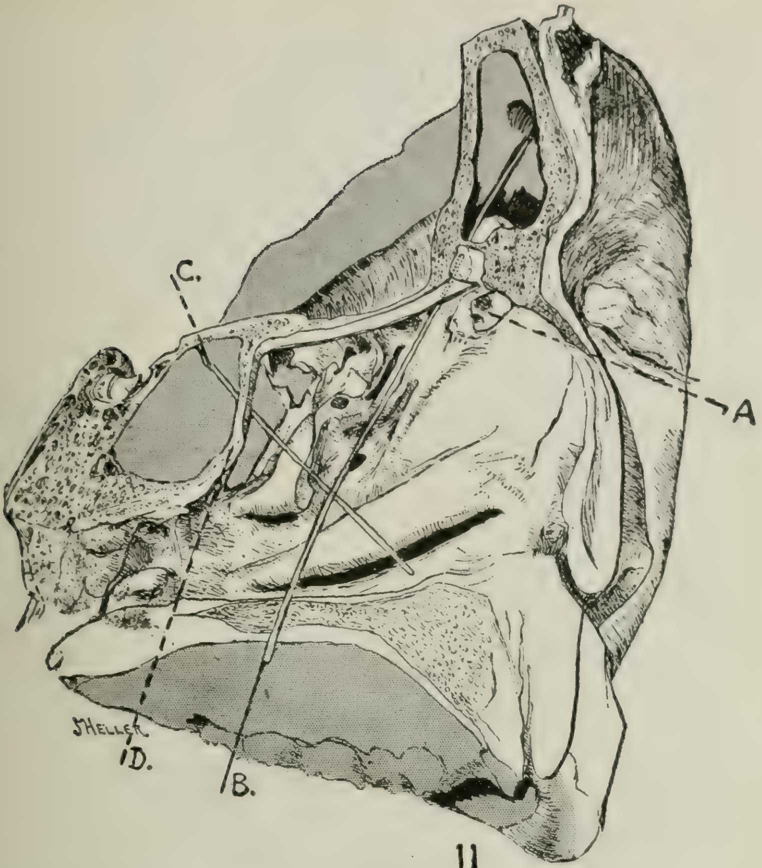

FIGURE 11.

Shows a three-quarter view of specimen with operation completed. The anterior half has been left "conservative"—i. e., the wall is intact, with all the openings free as far as one and one-half millimeters of the cribriform plate. An infundibular cell, A, has been opened, as shown first in Figure 5. The posterior half is "radical"—i. e., the anterior wall of the sphenoid cell has been removed with its entire postethmoidal part (lateral part, which is one-half or two-thirds of its extent). The postethmoidal cells have of necessity been dealt with radically to accomplish this—i. e., their entire nasal side has been removed. Bristle, B, is shown passing from the nose into the frontal sinus. Bristle, C, is shown passing through the postethmoidal-sphenoidal opening. A strip of bone, D, has been left to indicate the position of the sphenoidal-vomer junction

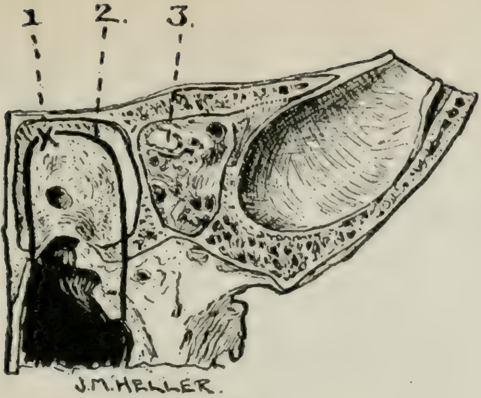

FIGURE 11A.

Shows diagrammatic representation of postethmoidal-sphenoidal cut shown in Figure 11. Transverse vertical section showing anterior sphenoidal wall viewed from behind. 1 the point X shows the entrance through the wall. The line 1-2 shows the extent of the incision. 3 the optic nerve.

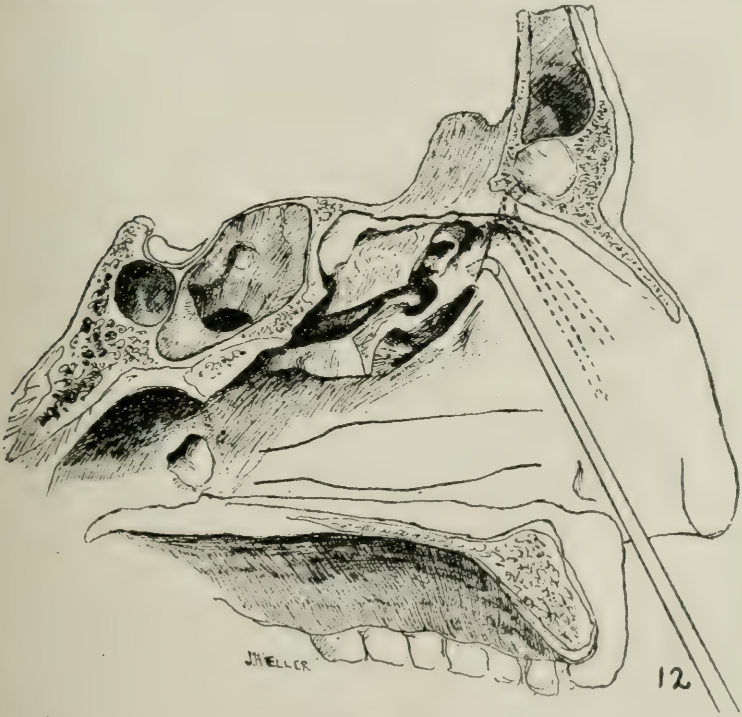

FIGURE 12.

Shows the knife in position for cutting away the uncinate process preparatory to Ingals' removal of the frontal sinus floor. Oftentimes the entire frontal floor may be cut away by the knife.

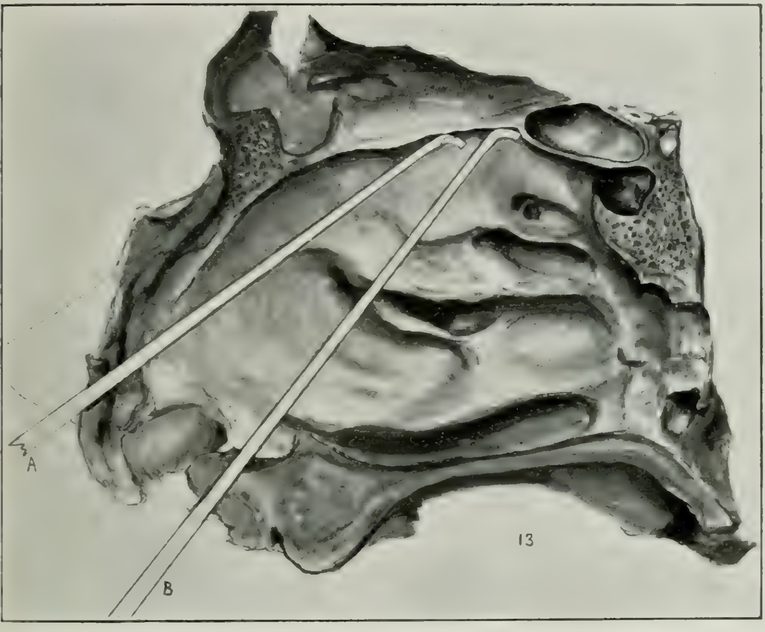

FIGURE 13.

Shows knife approaching a postethmoidal cell which lies on the top of the sphenoidal sinus and occupies about one-half of the body of the sphenoid. The line, A, shows position of approach. B shows the position for cutting. From considerable clinical experience I believe this cell would be opened by this technic.

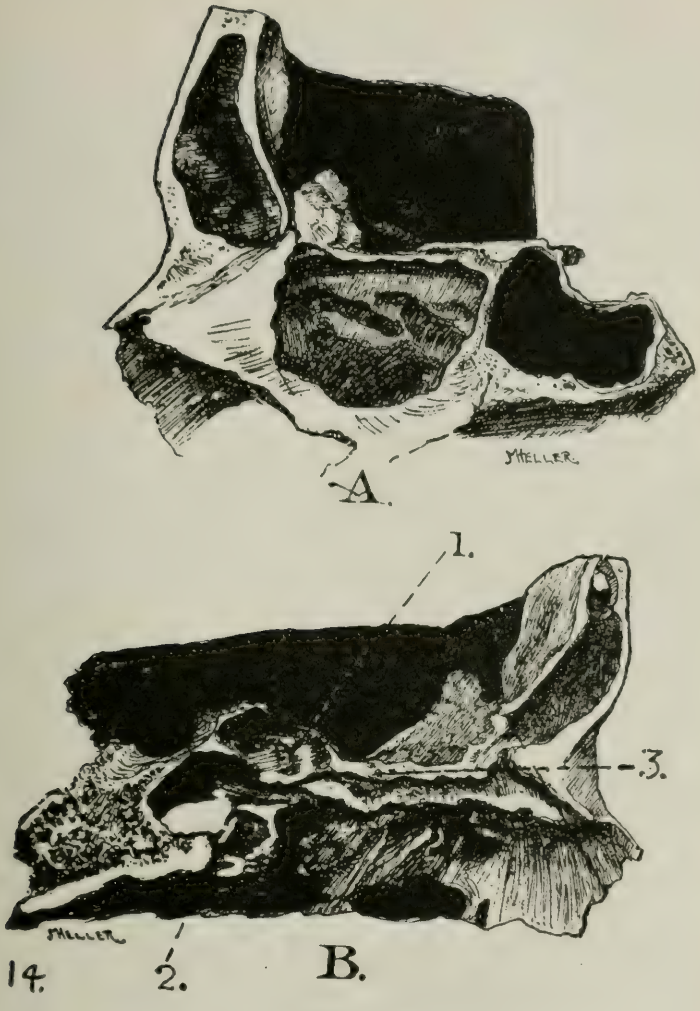

FIGURE 14.

A shows the usual sphenoidal sinus and the usual cribriform plate with their usual relations. B shows postethmoidal cell 1 placed altogether on top of sphenoidal sinus 2. Its lowermost limit is almost on the horizontal of the cribriform plate 3. This cell could not have been opened by this technic, nor, in my judgment, by any other extant.

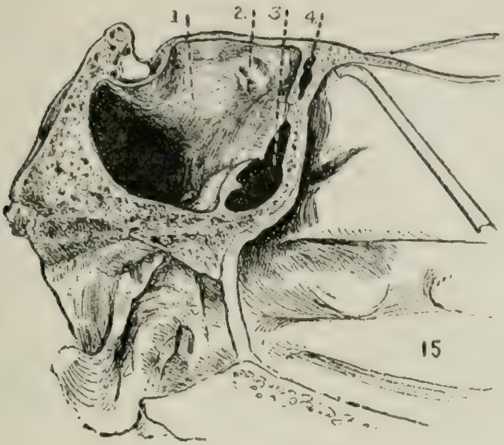

FIGURE 15.

Shows the left half of specimen. 1 right sphenoidal sinus extending into left side to border left optic canal 2. Left sphenodial sinus 3 also borders left optic canal. A postethmoidal cell, 4, also borders left optic canal. All three of these cells would probably have been opened by this technic operating on the left side.

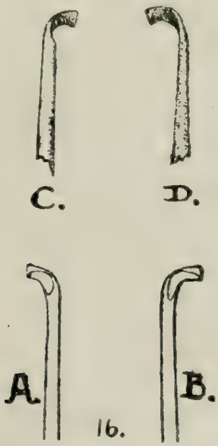

FIGURE 16.

A, sphenoid knife, B, turbinate knife. C and D, turbinate knives made right and left. Some of my confreres prefer right and left knives. I prefer the straight knife.

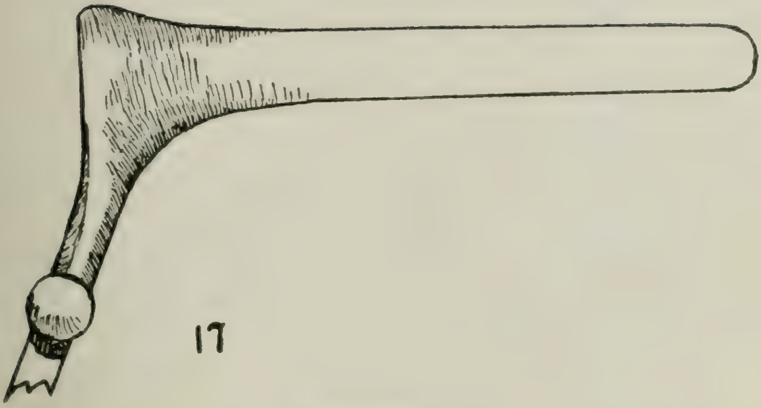

FIGURE 17.

Sphenoidal speculum. It is the primary bivalve nasal speculum made of tempered steel blades eighty-eight millimeters long, nine millimeters wide, knife edge thin near tip. It should have a set screw. It is a simple enlargement of the primary bivalve for this purpose, as Killian's enlargement is for the purposes of septum resection. It is thirteen millimeters longer and four millimeters wider than the usual model of Killian's. The sphenoidal knife used with this speculum is twenty-five millimeters longer than the regular knife. By means of this speculum the route to the sphenoid may be forcibly widened when it is too narrow.

XVIII.

ASTHMA ASSOCIATED WITH ETHMOIDAL DISEASE.*

By John Mackenzie Brown, M. D.,

Los Angeles.

The association of nasal diseases with asthma has been observed for many years, and especially the occurrence of polypi in these cases. There has been a great deal of work done recently in all departments of medicine, looking for local lesions which produce various chronic disorders throughout the body. It should be noted that the nasal lesions producing asthma are nearly all of ethmoidal origin, and that a more or less complete ethmoidal exenteration results in cure or in great improvement of the asthma.

According to Allbut, the pathologic anatomy of asthma is practically negative so far as the disease itself is concerned. Congestion, hyperplasia, and edema of the mucous membrane of the bronchi are frequently found, but he considers them either secondary to the repeated asthmatic attacks or due to an associated bronchitis. Williams and Longet first suggested that the actual spasm was produced by a contraction of the smooth muscle fibers which surround the bronchi. This was confirmed later by Hyde Salter in 1850, and worked out thoroughly by Brodie and Dixon in 1903. Allbut's explanation of the condition is the following: In moderate constriction of the bronchi, the powerful inspiratory efforts or movements are able to overcome the narrow tubes to a considerable degree. In expiration the forces which can be employed are much less efficient and are unable to expel the air, and the chest becomes fixed in a position of extreme distention. A very rapid swelling of the mucous and submucous tissues of the bronchi, due to vasomotor dilatation, was thought to be the cause of the asthmatic attacks, but it is now believed that the swelling of the mucous membrane is a secondary condition to the spasm of the bronchial musculature. The experiment of clamping the pulmonary vessels caused great engorgement of the bronchial mucous membrane, but did not produce any asthmatic symptoms. Horn, Freudenthal, and Keiper have observed in bronchoscopic examinations the presence of fibrous masses, ulcerations, and a general thickening of the mucous membrane. The accepted theory at present is that the symptoms are due to a spasm of the bronchial musculature, and that there undoubtedly is an edema of the mucous membrane, but that this is secondary. The question as to how this spasm is produced, whether by reflex or direct action on the muscle fibers by some substance in the circulation, has not been settled. A reflex necessitates action through the nervous system, while the direct action on the muscles could occur through the circulation, regardless of the nerves. The bronchial spasm has been attributed to the introduction of a foreign proteid to which a sensitization has been produced, or to a reflex irritation. Whether we accept the theory of hypersensitization or the reflex theory, there must be some other etiologic factor, because the same conditions in the nares present themselves in many cases, whereas only a comparative few develop asthma. Dundas Grant suggests that disturbance of the higher cerebral centers may interfere with the inhibition of reflex action in asthmatics. In our series of cases nearly all were of the neurotic type, and were so before the advent of any asthmatic symptoms.

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*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, White Sulphur Springs, May 5, 1916.

The reflex theory implies that these attacks are brought about by an irritant in the nares or at some other part of the body, thereby causing a reflex stimulation of the smooth muscle of the smaller bronchi. The irritation in the nose causing reflex action could come from contact of the ethmoid with the septum, as this is the most sensitive area, or the altered secretion may be the irritant. Every case in our series had a contact of some kind in the nares. The sensory fibers of the nasal mucous membrane are supplied by the fifth nerve, and, according to Landois, the origin of the sensory roots of the fifth nerve are connected with the motor roots of all the nerves arising in the medulla except the sixth nerve. Therefore, this connection accounts for the numerous and varied reflexes from irritation of certain branches of the fifth nerve. The tenth or vagus nerve arising from the medulla has intimate relationship with the nucleus of the fifth nerve, and supplies the motor fibers to the nonstriated muscles of the bronchi, or at least goes to form the anterior and pulmonary plexuses from which the nerves supplying these muscles originate.

The Anaphylactic Theory.—The experiments of Theobald Smith, Auer and Lewis, Otto, Meltzer, Matthews, Goodale, and others have shown that the injection of foreign proteids produce a sensitization of the body to that particular proteid, and that a subsequent injection of the same proteid produces the symptom complex known as anaphylactic shock, which has many characteristics of an attack of asthma. The phenomena exhibited in the bronchial tubes is undoubtedly, as the experiments of the above authors show, the same as in true asthma—i. e., spasmodic contraction of the involuntary muscles of the smaller bronchi. Just how the muscles are stimulated is unknown, whether it be due to stimulation by the toxin in the blood on this particular musculature, or by acting through the nervous system.

Auer and Lewis in their experiments obtained the same results in pitted as in unpitted animals, and the same symptoms of anaphylactic shock were observed after section of the vagi; these investigators will not, however, go so far as to state whether it is due to stimulation of the nerve terminals of the vagus or direct stimulation of the toxins on the musculature.

Matthews reports the same anaphylactic symptoms in animals in which the vagi had been severed and sufficient time had been allowed for complete degeneration of the peripheral fibers, as he obtained in animals in which the vagi had not been disturbed; he believes that the direct stimulation of the muscles through the blood is undoubtedly the cause of the spasm.

The investigations of Gay and Southard led them to believe the phenomena to be due to stimulation of the respiratory centers, causing violent inspiratory efforts of the diaphragm. The work of Auer and Lewis, and Matthews would seem to prove this to be erroneous.

Shultz has demonstrated that smooth muscle from a sensitized animal would contract more readily than from one non-sensitized.

In asthma due to pathologic lesions in the nose the absorption of pus or mucopus from these lesions is given as the cause of the attacks. Todd draws attention to the fact that sufferers from hay fever subsequently develop a distinct pathologic condition in the ethmoid cells; and that the asthmatic attacks in these cases may be due to inhaled pollen or to the ethmoidal diseases.

Koesler and Moody believe that bronchial asthma is due to a focal infection in the bronchi; that the subsequent bronchitis due to this infection is the primary cause of the asthma; and that the asthmatic patient has become sensitized to the infection causing the bronchitis. They found three anaerobic bacteria, two bacilli, and one streptococcus, in addition to the common aerobic bacteria. These bacteria, they believe, produce amines which cause the spasm of the muscles of the bronchi, and also cause ulcerated areas from which rapid absorption results.

In all our cases there was demonstrable some involvement of the ethmoids, and, no matter what other pathologic condition existed in the nose, this was a constant factor. Only five of our cases had suppurative ethmoiditis, and the probable reason that these cases do not result in asthma more frequently is that suppuration for any length of time will cause a degeneration of the normal mucous tissues and the nerve terminals.

There must be some underlying factor in these cases of ethmoiditis that will produce asthma, and this we believe to be the neurotic state of the individual, as we have all seen nares full of polypi with no asthmatic symptoms. We further believe that there must be asthmatic attacks due entirely to reflex action.

Mackenzie reports a case of a woman in whom he was able to produce repeated attacks of asthma by showing her an artificial rose. Chavonne reports a case of asthma of three years' duration due to a small papilloma of the uvula which was entirely cured by the removal of the tumor.

Two similar cases occurred in our practice. One, a woman whose asthma had been always relieved when she went to a certain resort. While enjoying perfect health at this resort she received a letter containing bad news, and was immediately seized with a violent asthmatic attack lasting for several hours. The other is a case with ethmoiditis, in which an attack of asthma occurs instantly upon going from a warm room to one several degrees colder.

Landois, by irritating the nasal branches of sphenopalatine nerve, produced asthmatic symptoms, and Brodie and Dixon were able to produce typical asthma, after decerebration, by stimulation of the nasal mucous membrane. This reflex was abolished by section of the vagi; and even if the spasm had already commenced, it relaxed at once on section of the vagi. They also proved that the upper parts of the nares were the most sensitive to stimulation.

Whatever the theoretical explanation may be, two facts have been observed for a long time by rhinologists: first, that the association of chronic ethmoiditis and asthma is marked; second, that great improvement in asthmatic symptoms follows removal of pathologic nasal lesions.

Our desire in this paper is to report observations on twenty-seven asthmatic cases in which exenteration of the diseased ethmoid labyrinth was done. It is worth noting, also, that these patients were great sufferers, and many of them had gone through the list of asthma remedies without relief other than temporary.

The ages of the patients in this series range from four years to sixty-two years.

Two were under ten years.
One, ten to twenty years.
Six, twenty to thirty years.
Eight, thirty to forty years.
Five, forty to fifty years.
Four were over fifty years.
Females, fourteen; males, thirteen.

There was a marked deviation of the septum in thirteen cases. The antrum was involved on one or both sides in three cases, the frontal sinus in two cases, and the sphenoid in two cases. The ethmoids were involved in every case; twenty-two were of the hypertrophic type, with no demonstrable pus, and five were suppurative.

The treatment in this series consisted in as near a complete exenteration of the ethmoids as possible, according to the method of Mosher. The septum was only operated upon when it was necessary in order to do a satisfactory ethmoid exenteration, or when the deviation would still cause some irritation in the nose after the exenteration. After an exenteration the deviated septum seldom interfered with respiration or drainage from the sinuses. The antrum was treated by the Krause operation, and it was noticed in two antrum cases that treatment directed to the antrum first, greatly lessened the ethmoidal lesions and also the asthma. The frontal and sphenoidal sinuses were treated at the same time as the ethmoidal exenteration, by simple enlargement of the frontonasal duct or the sphenoidal opening. In children tonsils and adenoids were removed.

THE RESULTS OF THE OPERATION.

Six were completely relieved, having no return of symptoms to date, the periods in which no attacks have occurred ranging from three to eighteen months after operative interference. Twelve were decidedly improved—that is, having only slight attacks at intervals, and these mostly during acute attacks of rhinitis or other physical ailments. Seven were but slightly improved, and two were not improved at all. In the latter two classes six had marked bronchial pathologic lesions.

We are convinced from our observations on these patients that hypertrophic ethmoiditis is a distinct etiologic factor in the production of asthma. The complete exenteration of the ethmoids offers the best results in chronic cases. We realize the great difficulty in doing a complete exenteration, as it is practically impossible to remove all the mucous membrane in the labyrinth. A small portion of the diseased mucous membrane seems to be able to continue the attacks. In asthma due to nasal lesions, when there has been a marked bronchial condition, the bronchial lesions will continue the asthma in spite of any operative procedures in the nose.

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No.	Age	Duration	Nasal Findings	General Findings	Operation	Time of Observation	Results
1	45	10 years	Septum deflected, hypertrophic ethmoiditis with many polypi. No pus.	Marked bronchitis, emphysema	Ethmoid exenteration.	3 months	Attacks lessened in number and severity.
2	40	3 years	Chronic suppuration of the ethmoids and antrae.	Moderate bronchitis.	Ethmoid exenteration.	4 months	Attacks lessened in number and severity.
3	43	5 years	Hypertrophic ethmoiditis. Middle turbinates almost filled the nose. No pus.	Moderate bronchitis.	Ethmoid exenteration.	8 months	Attacks lessened in number and severity.
4	54	6 years	Hypertrophic ethmoiditis with many polypi.	Moderate bronchitis.	Ethmoid exenteration.	12 months	Attacks lessened in number and severity.
5	41	6 mo.	Hypertrophic ethmoiditis with right nares full of polypi. No pus.	Negative.	Ethmoid exenteration.	3 months	Attacks lessened in number and severity.
6	38	6 years	Very narrow nares, hypertrophic ethmoiditis. No pus.	Marked bronchitis, emphysema	Ethmoid exenteration.	6 months	Patient notices no difference in number or severity of attacks.
7	28	12 years	Septum deflected, hypertrophic ethmoiditis with many polypi in right nares. No pus.	Moderate bronchitis, emphysema	Septum resection and ethmoid exenteration.	8 months	Attacks lessened in number but not in severity.

8	39	20 years	Septum deflected, hypertrophic ethmoiditis with very large middle turbinates. No pus.	Marked bronchitis, emphysema	Septum resection, ethmoid exenteration.	14 months	Attacks lessened in number and severity. Patient was unable to do any work for four months prior to operation. Since operation has been able to do housework.
9	49	6 years	Septum deflected, chronic suppuration of the ethmoids, antrae and right frontal.	Moderate bronchitis.	Septum resection, Krause on the antrae, ethmoid exenteration and right frontonasal duct.	8 months	Decided lessening of number and severity of attacks. Patient was unable to do any work prior to operation, but is now able to work half the time.
10	26	6 years	Septum deflected, hypertrophic ethmoiditis with many polypi. No pus.	Marked bronchitis, emphysema	Septum resected, ethmoid exenteration.	18 months	Decided lessening in number and severity of attacks.
11	30	2 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	18 months	Attacks gradually became less and entirely ceased nine months ago.
12	27	2 years	Hypertrophic ethmoiditis with many polypi. No pus.	Slight bronchitis.	Ethmoid exenteration.	10 months	The attacks entirely disappeared six weeks after operation.
13	29	7 years	Hypertrophic ethmoiditis, many polypi, very narrow nares. No pus.	Chronic bronchitis, emphysema, chronic pulmonary tuberculosis.	Ethmoid exenteration.	14 months	Decided lessening in number and severity of attacks.

No.	Age	Duration	Nasal Findings	General Findings	Operation	Time of Observation	Results
14	38	8 years	Hypertrophic ethmoiditis. No pus.	Moderate bronchitis.	Middle turbinates removed 3 years ago, ethmoid exenteration two years later.	3 years	After removal of middle turbinates, patient was completely relieved for two years, when asthma again developed. After exenteration, no attacks.
15	13	6 years	Deviated septum, hypertrophic ethmoiditis, middle turbinates, hypertrophic tonsils and large adenoids. No pus.	Moderate bronchitis.	Septum resection, tonsils and adenoids removed.	6 months	No asthma since operation.
16	62	17 years	Septum deflected, hypertrophic ethmoiditis with polypi. Slight pus discharge from both nares. After ethmoid operation pus was discovered in right sphenoid.	Marked bronchitis, emphysema	Septum resection, ethmoid exenteration.	4 years	Attacks decidedly lessened in number and severity.
17	53	6 years	Hypertrophic ethmoiditis. No pus.	Marked bronchitis, emphysema	Ethmoid exenteration.	10 months	Attacks lessened in number and severity.
18	35	3 years	Hypertrophic ethmoiditis with many polypi. No pus.	Slight bronchitis.	Ethmoid exenteration.	10 months	No attacks since operation.
19	52	5 years	Septum deflected, hypertrophic ethmoiditis.	Slight bronchitis.	Right ethmoid exenteration.	5 months	No attacks since operation.
20	30	3 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	8 months	Decided lessening in attacks in number and severity.

21	30	1 year	Suppuration of ethmoids, antrae, left frontal and left sphenoid.	Negative.	Ethmoid exenteration, enlargement of frontonasal duct and sphenoidal opening. Krause on antrae.	11 months.	Patient has had one attack since operation, due to an acute rhinitis, and several mild attacks after exposure to severe weather.
22	35	4 years	Hypertrophic ethmoiditis with polypi. No pus.	Slight bronchitis.	Ethmoid exenteration.	22 months	There has been no attacks for the past fourteen months.
23	20	3 years	Septum deflected, hypertrophic ethmoiditis with very large middle turbinates. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	12 months	No asthma since operation.
24	4	1 year	Hypertrophic ethmoiditis, hypertrophy of tonsils, and large adenoid.	Negative.	Adenoid and tonsils removed.	15 months	Attacks lessened in number and severity.
25	4½	1½ years	Hypertrophic ethmoiditis, hypertrophy of tonsils, and large adenoid.	Negative.	Adenoid and tonsils removed.	9 months	No asthma since operation.
26	28	7 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Ethmoid exenteration.	10 months	No asthma since operation.
27	25	2 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	9 months	Attacks lessened in number and severity. Slight attacks during acute rhinitis, and whenever he has digestive disturbances.

XIX.

REPORT OF THREE CASES OF BRAIN ABSCESS OF OTITIC ORIGIN.*

By George M. Coates, M. D.,

Philadelphia.

Case 1.—M. O., aged fifty-one years, a waiter, came to the Out-Patient Department of the Pennsylvania Hospital, July 6, 1912, complaining of pain in the right ear of two days' duration. He gave a history of having had a right sided aural discharge for several days four months previously, but with no discomfort in the interim. Examination showed a bulging membrane, which was at once incised in the posterior portion of the membrana, a somewhat scanty discharge resulting. Some mastoid tenderness was present which became slightly more marked in the next few days, and he was admitted to the ward on July 9th. He did not complain particularly of headache. No signs of meningitis were present, and the leucocyte count was practically normal. The patient was a well developed and well nourished man. Eyes showed no spontaneous nystagmus, pupils were equal and reacted to light and accommodation, eye grounds negative. There was no aphasia. The teeth were in bad condition; throat normal and tongue slightly coated. Heart, lungs and extremities normal. Urine normal. Temperature, 99°; pulse, 88; respirations slightly high.

Expectant treatment was carried out for a few days, as the temperature continued low, but some slight swelling appearing over the mastoid, he was operated upon on July 15th. The mastoid cells were all found normal with the exception of the antrum, the tegmen of which appeared soft, if not necrotic, this softened area extending outward into the temporal ridge. The dura was exposed for inspection and appeared to be under considerable pressure, so it was incised and the brain searched with a thin knife blade. In a direction inward and upward, about an inch from the surface, an abscess cavity containing about two drams of pus was encountered. It appeared to have rather firm walls. Gentle washing cleaned out the pus; a cigarette drain was inserted, and the lower portion of the skin wound closed. Cultures from the pus failed to show any growth. Recovery was slow but perfectly uneventful, the abscess continuing to drain nicely, although the middle ear ceased at once to discharge. The drain was removed in about ten days and the wound allowed to close by granulation, which it did completley in about six weeks. During this time the temperature never rose above 99 4/5°, being normal for the most part, the pulse ranging from 64 to 88. Respirations were always a little rapid.

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*Read at the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, held at Philadelphia, March 3, 1917.

This case showed nothing clinically to suggest brain abscess, the bulging of the brain and dura through the operative bone wound being the only indication which led to its discovery. As the contained pus was apparently sterile, and as the cavity had definite walls, it is probable that it had its inception at the time of the first ear trouble, four months earlier. It illustrates very well the type of abscess that is favorable to recovery, namely, a walled-off, chronic process with sterile pus, or at least having organisms of low vitality, discovered at the time of operation without having given rise to pressure symptoms.

Case 2.—C. C., twelve years old, school boy, admitted to the Pennsylvania Hospital May 24, 1913. Temperature, 99°; pulse, 100; respirations, 24. He had had a simple mastoidectomy on the right side two years before, followed by a radical on the left side one year later. On admission he gave a history of pain in the right ear for five days, accompanied by a free discharge of pus. For two days past there had been marked general headache. He was a frail, undersized boy, irrational, whining, with considerable photophobia. The eyes reacted well to light and accommodation; grounds apparently normal. Heart and lungs were negative. The neck was quite rigid, suggestive Kernig signs were present on both sides and a doubtful Babinski; reflexes not markedly increased. There was swelling and redness over the right mastoid region, which was markedly tender. The right membrana showed a large central perforation in a thickened drum, freely discharging foul pus with a distinct musty odor. The left ear, on which a radical operation had been performed one year before, was not entirely dry. The leucocyte count was 21,700, with a high percentage of polymorphonuclears. By lumbar puncture the spinal fluid was found to be cloudy and under considerable tension, with the polymorphonuclears sixty-seven per cent. No tubercle bacilli were found, and no growth was obtained on culture media.

Although all his symptoms were considerably relieved by the lumbar puncture, he was operated upon by Dr. Francis R. Packard, on whose service he was, and much destruction of bone in the upper portion of the mastoid found, extending into the temporal ridge and zygomatic cells. The dura was freely exposed and the wound packed with iodoform gauze. Whereas he had been markedly restless, delirious and noisy previous to operation, he now became much quieter and mentally more clear. Very foul pus continued to drain from the wound, and at this stage Dr. Packard went off duty and he came into my hands. The improvement following the first operation was not permanent, however, and during the next few days all of his former symptoms returned in greater force, the neck being well arched backward and his delirium increasing. On July 9th, ten days after the first operation, in dressing the wound, I noticed that the dura was tense and bulging in the bony opening through the site of the temporal ridge, where the removed bone covered an area about three by four centimeters. A paralysis of the external rectus muscle of the right eye was now apparent. The pus was very foul in odor. With very light anesthesia, I incised the dura and searched the underlying brain with a grooved director, when a large abscess cavity was discovered containing about an ounce of thin, foul pus. A rubber drain was inserted without washing and the wound dressed. The patient was very weak and partly comatose. The pus showed the colon bacillus in pure culture, and continued to drain very freely, and always foul, until, gradually sinking, he died five days later. At this time there was considerable protrusion of the brain tissue into the wound, the protruded portion being necrotic. The temperature had ranged from 99° on admission to 104° on May 26th, where it stayed for two days. It dropped to 99° for a few days and again rose to 104 3/5° when the second operation was performed. Thereafter 103° was the lowest temperature until death. The pulse rate was commensurate with the temperature, running from 100 on admission to 160 shortly before death. Respirations were always rapid—from 24 to 32, and 52 at death. No autopsy was obtainable, but from the appearances presented, I think it probable that there was a second abscess that was not evacuated. The patient's condition while upon the operating table was so grave that I did not feel inclined to search further after draining the abscess.

Case 3.—Laura H., thirty years old, stenographer, was admitted to the Pennsylvania Hospital July 6, 1916, complaining of pain in the left ear, left sided headache and general malaise. Temperature, 102°; pulse, 92, and respirations, 24. Family history was without interest. She had had measles in childhood, which did not affect the ears as far as she could remember; had always had "catarrh," indigestion and constipation. The left ear had suppurated, with severe earache, fifteen years previously, and had ceased discharging after a few days. During the next few years she had had several similar attacks in the left ear, and finally the discharge had been almost continuous for the past six years, although always scanty. She had not had the ear treated, considering it a negligible matter, except for the impaired hearing.

The present illness dated back six days, when she developed an acute rhinitis from sitting in front of an electric fan while at work. Earache immediately followed and she went to bed. There was an attack of vomiting the same evening, and as she felt badly she was admitted to another hospital the next day, July 4th. Not getting any better here, and having almost continuous nausea and vomiting, she left this institution and was admitted to the Pennsylvania Hospital, where the drum was incised at once. Temperature on admission was 102°; pulse, 92; respirations, 24. She was a well developed and well nourished, full faced, rather pale adult, complaining bitterly of pain in the left ear. There was no adenopathy, the eyes were slightly prominent, pupils widely dilated, equal, reacted normally to light, grounds normal. The nasal septum was deviated to the left, the teeth in fair condition, tongue coated, throat negative. A scanty discharge came from the left ear, and there was no retraction of the head, although slight pain on extreme flexion of the neck and a suggestion of Kernig's sign in the right leg. Reflexes normal. No spontaneous nystagmus was present, vestibular reaction to the caloric test normal. Widal and Wassermann reactions negative. The blood count showed a leucocytosis of 14,000, polymorphonuclears seventy per cent. Urine negative. Patient was very uncomfortable. No aphasia—mentally slightly sluggish.

On July 8th complained of considerable pain in the ear and back of the head, as well as in the nose. There was very little aural discharge. The eye examination by Dr. Wm. T. Shoemaker follows: Externally nothing noticeably abnormal. Pupils large and equal. Irides react to light. Ocular movements full. Eye grounds: media clear, discs well defined, vessels good, no hemorrhage or exudation or any evidence of intracranial trouble. Temperature, 99°; pulse, 80; respirations, 20. Her condition continued no worse for two days, when the temperature rose to 102°, pulse 84, respirations 20, and she vomited a grassy green substance. Pain was more intense on the vertex and occiput; there was slight rigidity of the neck, and Kernig's sign was positive in the right leg. Dr. Morris J. Lewis, the internist, confirmed the diagnosis of meningitis. The mastoid was not markedly tender, and there was no sagging of the posterosuperior canal wall. By lumbar puncture thirty-five cubic centimeters of fluid under pressure were withdrawn. Fluid was cloudy, there was slight reduction of Fehling's solution, cytologic count was one hundred and eighty-six, polymorphonuclears eighty per cent. The blood culture was negative. As the patient was much more comfortable following this, it was repeated on the following day, July 11th, and twenty cubic centimeters of cloudy fluid, not under pressure, were withdrawn, with apparently increased comfort. At midnight, however, she again complained of great pain, which morphin failed to control. She became weak and cyanotic during the remainder of the night and early morning. The pulse was very weak and the temperature went to 104 1/5°; respirations, 24. The report of the X-ray picture, taken July 10th, proved to be very misleading, as follows: "Extensive necrosis of the entire cell area of the left mastoid, sinus probably uncovered. No further evidence as to pressure or location of any suspected brain abscess."

The left mastoid was opened July 12th and found to be eburnated, without cells or necrosis, solid and intact; the kind frequently seen accompanying prolonged middle ear suppuration. Believing that we were dealing with a case of purulent meningitis with the possibility of brain abscess, I freely exposed and incised the dura of the temporal lobe, obtaining a little cloudy cerebrospinal fluid. The tension of the brain appeared to me to be increased, so I searched it cautiously with a grooved director and tapped an abscess cavity containing nearly an ounce of thick pus. Up to this time the patient's condition had been precarious. The temperature was 102°; pulse, 80, and respirations, 28, just before being placed on the table; she was comatose, cyanotic and breathing was slightly stertorous. The pulse was very weak. Very little ether was used in anesthetization. After draining the abscess, almost immediately her pulse improved, her color became normal, and her breathing was easier. A rubber drain was inserted, the wound packed and dressed, and she was sent to the recovery ward, by this time already conscious and apparently rational. Before I had finished dressing, however, word came that she was sinking rapidly, and when I reached her a moment later she was cyanotic, although the pulse was fair and continued so for several moments. Respiration became weak and ceased. Artificial respiration and stimulants were of no avail, and she died of respiratory failure.

The autopsy diagnosis was multiple (two) brain abscess in the left temporal lobe, purulent meningitis. The complete autopsy report follows:

"There is an operation wound with a drain inserted reaching into the cranial cavity behind the left ear. There is a slight clouding beneath the pia. The various arachnoid spaces contain cloudy fluid. The fluid in both ventricles is purulent—less so in the right. The right cerebrum is otherwise normal. The drain reaches into an abscess cavity within the temporal lobe, about three centimeters posterior to its anterior end and opening in about the line of the inferior temporal sulcus. This cavity is just beneath the surface, measures about two centimeters in diameter, contains some purulent fluid and has a soft, thin wall of necrotic cerebral tissue. There is also another abscess which opens into the posterior descending horn of the left ventricle and to the surface proximal to the cerebellum at about the posterior third of the collateral fissure and slightly lateral to that in the fusiform gyrus. This abscess cavity is the larger of the two, in that its openings in both directions are large. There is about one centimeter of cerebral tissue in between the two cavities.

Microscopic Examination.—The edge of the brain cavity is densely infiltrated with leucocytes and to a less extent with plasma cells. There is no fibrous reaction about the inflammatory area. The cavity is not of long standing."

From the autopsy report these abscess cavities seem to have been of recent formation, and there is nothing in the history to indicate prolonged intracranial involvement except the duration of the aural discharge—fifteen years. Of course, we know that a brain abscess frequently lies dormant for long periods of time to eventually become active and dangerous under some fresh stimulation, such as the chilling from the electric fan and consequent aural reinfection in this case. Although we suspected brain abscess almost from the start from the character of the headache, nothing confirmatory of this could be discovered, and the development of the meningitis was so evident, that that exclusive diagnosis was made. I could not be quite content with this, however, or feel that it accounted for all of her symptoms, and therefore searched the brain at the time of operation, with the discovery of the first abscess. I did not suspect the second one. Immediately following the evacuation of the first abscess her condition was in all respects so satisfactory that I feel I was justified in believing the source of the trouble had been discovered and eradicated, and I sent her away from the table feeling that recovery was not at all to be despaired of. Whether the sudden collapse that followed within ten minutes was due to the rupture of the deeper abscess into the ventricle I do not, of course, know, but think that is probably what occurred, since such a rupture was discovered at autopsy. Or it is possible that there had been a slow leakage there which accounted for the infection of the cerebrospinal fluid. There was no "stalk" or ascertainable avenue of infection to or from the middle ear, although this must undoubtedly have been the origin of the brain lesion.

These three cases only serve to emphasize still more the well established dictum that a chronic discharging ear, no matter how trivial it may appear, or how quiescent during long periods of time, is always a serious menace to the life of the patient. No one fact pertaining to suppurative otitis media is, perhaps, better established, and yet how many such middle ear conditions are neglected and temporized with. J. W. Murphy lays stress upon this point in his article read before the society in June, 1915, and considers cerebral abscess of otitic origin almost always an acute process, and that, therefore, eye pressure symptoms have not time to develop, as in the more slowly growing tumors. In only one of my cases was there any paralysis apparent—that of one of the eye muscles in the second case—and this came on late in the disease. Neither were the mental symptoms more conclusive or helpful in establishing the diagnosis. In one of these cases, the one that recovered, there was little elevation of temperature, but in the other two the febrile reaction was marked—none was ever subnormal. The pulse rate was rapid, at least commensurate with the temperature, as were also the respirations. With wakefulness and delirium rather than slow cerebration, and mental alertness instead of drowsiness, the absence of eye pressure symptoms or of paralysis, and in two of the three cases a concomitant meningitis existing, the diagnosis of a brain abscess was a difficult task. As a matter of fact, it was only made on the operating table, after a search of the brain on suspicion, in two of the cases.

XX.

SEVERE EPISTAXIS IN A WOMAN WITH DIABETES, MYOMATA OF THE UTERUS AND ACUTE MASTOIDITIS.*

By Wolff Freudenthal, M. D.,

New York.

On account of its numerous complications, the following case may be of interest to the Fellows of this section:

Mrs. ———, aged about forty-five years, called on me in the middle of the night of January 16, 1917. She was accompanied by two physicians, who had been unable to stop the severe epistaxis from which she had suffered for several hours. The bleeding had started in a mild form at noon; a physician was called in and left a prescription for adrenalin with instructions; but the hemorrhage continued and became profuse during the evening, when the two other colleagues were summoned. When I saw the patient she showed some signs of acute anemia. With some difficulty the bleeding was finally stopped by plugging the nose anteriorly. Adrenalin, it was soon found out, had here the opposite effect to what it generally has—it increased the hemorrhage.

As this lady was of an age when menopause sometimes occurs, and having encountered numerous instances of vicarious menstruation, I inquired about the menses, and was told that she was just having them, that they occurred every three weeks and lasted for eight days or more. Her family physician, furthermore, stated that she was suffering from multiple myomata of the uterus, the removal of which before the next period I strongly advocated, in order to avoid further trouble from the nose.

The patient was taken home and rest and ice applications ordered. The next afternoon I visited her and removed the gauze, but had to pack again on account of the occurrence of quite some bleeding. During the following night a severe hemorrhage set in, which was arrested by my assistant. The next morning (January 18th) she complained of toothache, having had for some time past several carious teeth, one of which had to be extracted a week before. Undoubtedly the toothache was caused by the ice applications, which now had to be discontinued, this complicating somewhat the treatment of the epistaxis. Besides, the question of her having another tooth extracted came up, but it was deemed inadvisable to do so on account of the continued epistaxis, which, I believed, would not cease until her menses were over. Such was the case, for the bleeding stopped entirely on January 22d. Before that happened, however, an otitis media set in on January 19th, so that a paracentesis had to be made, with relief of a good deal of the pain from that source.

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*Read before the New York Academy of Medicine, Section on Laryngology, March 28, 1917.

January 25th, on examining the nose and throat, I was struck by the peculiar aspect of the pharynx. The nose did not present any peculiar feature except that the mucosa was very dry, and anteriorly on the septum, at the socalled locus Kiesselbachii, there was the place from which the bleeding had started. The pharynx, however, was different. The entire mucous membrane was also very dry. The velum and its vicinity was somewhat edematous, and on the wall of the pharynx there was seen a distinct superficial ulceration. No lues nor tuberculosis being present, I had the urine examined, and it was found to contain four and four-tenths per cent of sugar plus acetone.

Naturally that upset our plans as to the removal of the myomata of the uterus, and it was thought best to resort to applications of Roentgen rays instead, which, by the way, had given very good results so far. In our specialty, it seems to me, we ought to make more use of those rays than we do. But that only apropos.

As soon as the patient was able, two teeth were extracted under cocain and the dental neuralgia arrested.

In the meanwhile, however, her otitis media had commenced to trouble her. A profuse purulent discharge, pain and swelling over the mastoid—in short, positive signs of a mastoid involvement—appeared. But I hesitated to operate, as the sugar had gone up to five per cent and the patient was in nowise a good surgical risk. As soon as she was somewhat stronger, and when the sugar had come down to three per cent, I found it necessary to operate, being well aware of the chances I took. The result was good beyond our expectation. She stood the narcosis (gas oxygen) very well, and healing of the wound was uneventful.

In going over this case it will be noted that there are two points of special interest. The first is that the possibility of diabetes being present was first suggested by a laryngologist. When I had a chance to examine the pharynx thoroughly, its peculiar appearance made me suspect diabetes. Years ago I described diabetic ulcerations of the pharynx and larynx,[1] but, with few exceptions, my reports were not noticed. Ever since I had occasion to see such cases occasionally, but did not collect them for publication, in the belief that the occurrence of such lesions had been established firmly by me.

Lately, Arrowsmith[2] published the brief history of a case of diffuse diabetic ulcerations of the pharynx and larynx, which would make, as he adds, "the seventh case of diabetic ulceration of the upper air passages so far recorded—the sixth, unless further search reveals Westbrook's observation." I too have searched for Westbrook's publication, but have been unable to find a single mention of it in the literature, either before or after my publication (sc. of five cases). The only one who had noticed something similar, namely, an abrasion on a vocal cord in a diabetic patient, was Edward Law of London. But that was only a side issue, to which he paid no attention. The main feature in his case was a subglottic nodule of doubtful origin.[3]

When I first described such cases, I thought to be justified in drawing the distinction between benign and malignant ulcerations. I am still of the same opinion. Whether the character of the ulcer depends upon the underlying disease, diabetes, as seems natural to suppose, or upon local conditions, is impossible to state. Our patient certainly belongs to the class that heals easily under general and local treatment. I am almost inclined to believe that in some, perhaps in many, instances the ulcers heal without any local applications. Producing no symptoms (especially no dysphagia), they are so often overlooked.

The second point of interest in my case was that a mastoid operation had to be done on a patient affected with diabetes. That such a complication is a grave one, especially in a subject still suffering from an acute anemia, nobody can deny. Such an operation is a far more serious affair than ordinarily, and any surgical intervention under such circumstances may have a fatal outcome. Still, no other choice was left than to open the mastoid and give free exit to the pus collection. It may be added that the preoperative mental condition of the patient was all that could be desired. She did not know that she was suffering from diabetes, nor from a mastoid. We told her that an abscess had to be opened, and she did not inquire for more details. I consider that psychical condition a very important factor in major operations, and the more we learn to act accordingly, the better it will be for the patient. As to the operation, no stress was laid on elegance nor thoroughness; free drainage had to be established in as short a time as possible. And that was done. The patient slept almost constantly for twenty-four hours after the operation; one-fourth grain of morphin had been given before. The final outcome was extremely gratifying.

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XXI.

SEPTAL HEMORRHAGE—ITS CURE BY SUBMUCOUS ELEVATION.

By John Leshure, M. D.,

New York.

The steps of this procedure are:

1. Induction of analgesoischemia (this term is suggested for the state which follows the local application of cocain and epinephrin or their substitutes, and its adoption is urged as being more comprehensive than the terms generally in use).
2. Incision and elevation of the septal mucoperichondrium backward for one inch and down to the floor of the nose.
3. Compression of the elevated membrane between the blades of a special forceps.
4. Replacement of the flap and packing in the usual manner for from twelve to twenty-four hours.

Indications for operating:

1. Cases of septal hemorrhage which resist ordinary methods of treatment, such as cauterization, astringents, etc., and those in which no bleeding point can be readily found.
2. Cases of chronic crusting of the septum accompanied by bleeding.
3. Cases of septal hemorrhage of mild or severe degree in children who will not tolerate local treatment and who are best operated upon under general anesthesia.

The principle of cure is obviously the destruction of the thin walled superficially placed vessels which bleed at the slightest provocation, and the ability to grasp the vessel between the blades of a forceps constitutes a distinct advantage, since pressure crushes the intima coat of the vessel, and obliteration of its lumen is thus accomplished.

The bleeding points are usually low down and anterior, consequently the incision should be placed as far forward as possible, and the elevation carried well down to the nasal floor.

If it is found difficult to reach with the compression forceps the vessels near the floor of the nose, a horizontal incision should be made along the floor joining the lower end of the vertical incision, or, in other words, one makes an L-shaped flap, all parts of which can readily be compressed. These two incisions sever the continuity of the vessels passing through Stenson's canal and those which pass into the mucous membrane from the maxillary crest.

Some of the vessels are of considerable size, and their anastomosis is so free that cauterization often fails to accomplish a cure.

In cases of chronic crusting accompanied by bleeding, the membrane is bound down by fibrous bands which interfere with the circulation and cause distention of the vessels at one point and constriction at another.

When these bands are divided by the elevation the blood supply readjusts itself, becoming more nearly normal, and the general tone of the membrane is decidedly improved, with a lessened tendency toward crusting.

This improvement has been noted after the submucous resection, both by the writer and others, and will be referred to later.

It is easier to operate between attacks, but if other means fail, one can usually check the bleeding in the following manner and operate immediately:

A pledget of gauze soaked in a ten per cent solution of cocain in 1:1000 adrenalin is placed in the bleeding nostril while the opposite septal surface is painted with cocain-adrenalin syrup (fifty-five per cent strength), the object being to secure a reflex vasomotor constriction of all the neighboring vessels.

If oozing still continues, the bleeding side may be packed firmly with oily gauze; which will allow but little blood to escape, and while still in situ, incision and elevation underneath the packing may be carried out. Unless the case is one of marked hemophilia, no further difficulty will be encountered.

Overaction of the heart, due to extreme nervousness, is apt to cause continued hemorrhage, and a hypodermic of morphin and adrenalin in proper proportions will check the bleeding and quiet the patient's fears.

A good balance is one-sixth grain of morphin sulphate dissolved in ten minims of 1:1000 solution of adrenalin. If more adrenalin is used, the morphin should be increased to prevent excessive rise in blood pressure.

Morphin dilates the medium sized vessels, at the same time slowing the action of the heart, thus lowering general blood pressure.

Adrenalin increases the force and frequency of the heart beat, and this action, added to the contraction of the peripheral vessels, raises the pressure.

By giving the drugs together the desired effect of both is obtained—viz., lowered blood pressure and contraction of all of the small blood vessels—owing to the predominant action of the morphin.

Children and nervous adults may be given a general anesthetic if necessary, and the anesthesia may be limited to the primary stage if the septum is painted with cocain-adrenalin syrup.

Shock and bleeding are considerably lessened when local analgesoischemia is added to the general anesthesia.

Ligation of the bleeding vessels has been suggested by some writers, but the membrane is usually so friable that such a proceeding must be attended with considerable difficulty.

A smooth flat-bladed angular forceps has proved most convenient for compressing the membrane, but an ordinary dressing forceps answers almost equally well. Care should be taken not to use sufficient pressure to cause necrosis and sloughing.

The wound edges may be brought together with sutures if one cares to spend the time upon this refinement of technic. Usually healing occurs promptly upon replacement of the flap, and in the writer's series there has been no instance of perforation or other untoward result.

A convenient method of packing is with strips of oily gauze introduced in successive layers with a piece of rubber tissue placed next to the wound.

If packed in this manner the strips may be removed in reverse order and without danger of disturbing the wound edges.

The after-treatment is that usually given resection cases—simple cleansing and protection of the wound with boric ointment or some similar preparation.

Dr. J. A. Thompson[1] and Dr. Joseph Prenn[2] both cite cases of septal hemorrhage cured by submucous resection of the septum.

The writer's experience, however, based upon forty-one cases of submucous elevation with compression, leads him to believe that resection of the cartilage is unnecessary, unless, of course, indicated because of nasal obstruction, or for other reasons; in other words, elevation and compression of the mucoperichondrium will cure practically all cases of septal hemorrhage.

In the opinion of many rhinologists, resection of the septum should not be undertaken when the patient is less than sixteen years of age, and since many of our cases of epistaxis occur in children, a method which will not interfere with septal development is preferable.

In the transactions of the 1916 meeting of the Oto-Laryngological Section of the American Medical Association there is printed a paper by Dr. Chester C. Cott of Buffalo, entitled "Xanthosis and Other Septal Hemorrhages."

The treatment is somewhat similar to the one just described, and consists of incision and elevation of the mucoperichondrium, followed by the introduction of iodoform gauze, which is snugly packed between the septum and the perichondrium. This is left in position for two days and replaced if bleeding follows its removal.

He reports thirty-eight cases, all of which were ultimately cured.

The introduction of packing between the septum and elevated flap seems to us unnecessary, and the possibility of causing recurrence of the hemorrhage when the packing is removed would seem to be a disadvantage.

Most of the present writer's cases are private patients, and have been seen from time to time during the past three years. The first private case was operated upon December 4, 1913, although several cases had been previously operated upon during the earlier part of 1913, at the Vanderbilt Clinic.

All of the patients have been free from recurrence of hemorrhage except one, a woman aged seventy-five years, whose blood pressure was two hundred and thirty at the time of operation.

The bleeding was checked at the time, but recurred six months later. Reoperation resulted in permanent cure.

The question often arises whether it is advisable to check a moderate degree of bleeding in cases of this kind. The patient insisted upon having relief from the inconvenience of repeated attacks of nose bleed, although probably these were merely physiologic.

One of the series was a case of acute hemophilia, possibly due to pyorrhea alveolaris.

The patient was a stock broker, aged forty-eight years, somewhat alcoholic and otherwise careless of hygiene, although his blood pressure was not excessively high.

Elevation and compression checked the bleeding from the anterior portion of the septum, but two days later blood appeared in the pharynx, and a bleeding point was discovered in the posterior portion of the inferior turbinate. Fresh blood also appeared in the stools, and evidently came from the lower bowel. He was given several injections of human blood serum by Dr. J. E. Welch of this city, and the hemorrhage ceased, although it was deemed advisable to keep the nose packed for about two weeks.

Later on the pyorrhea was treated, and up to the present time, a period of about one year, there has been no recurrence of bleeding.

Several of the writer's colleagues at the Vanderbilt Clinic have successfully used this method of treating septal hemorrhage, and have been able to verify the permanence of the cure in all cases which could be followed up.

SUMMARY.

The object attained by incision and elevation of the mucoperichondrium is free surgical access to the bleeding area, and the rational treatment of a bleeding vessel is compression, causing obliteration of its lumen.

Compresson en masse with a flat bladed forceps reaches all parts of the membrane, and if thoroughly done results in permanent cure.

It is one of the most satisfactory methods of managing cases of septal hemorrhage in children, since the operative treatment is completed at one sitting, and no prolonged after-treatment is required.

Although the number of cases reported is comparatively small, the evidence adduced by the writer and others is so favorable to this method of operation that it is confidently recommended as a routine procedure in appropriate cases.

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XXII.

SOME INTERESTING LABYRINTH AND EIGHTH NERVE CASES.*

By H. B. Graham, M. D.,

San Francisco.

We have all of us in our examinations of the eighth nerve neuritis cases been struck with the multiplicity in the causes and in the variation in the symptoms. The diseases of all the cranial nerves seem to follow about the same course, exhibiting symptoms and ability to recovery varying according to the function of the nerve, to the quality of the cause of the lesion, and to the intensity of the attack. I have in a former publication pointed out these qualities in respect to the sixth nerve, which have been so ably reviewed by Passow and others, and they have been referred to in the description of the peculiar menieriform polyneuritis of the cerebral nerves described by Frankel Hochwart. For the sixth nerve the causes have been proven by autopsy to be: First, pressure, as from a tumor; second, serous meningitis from middle ear infection; third, the effect of suppurative processes at the apex of the petrous portion of the temporal bone; fourth, toxic, as from lues, lead, arsenic, alcohol and tobacco, or from intestinal toxemia. We can readily see how these same causes might act on any cranial nerve, and especially on the eighth, but in addition to these we have arteriosclerosis, heart lesions, and a nerve exhaustion appearing as prominent factors with the eighth, factors which have not been mentioned in other cranial nerve neuritides. The eighth being so peculiarly mixed in its function, and so intimately connected with other portions of the nervous system, exhibits more pronounced susceptibility to remote influences and shows a larger variety of easily demonstrable symptoms than do other cranial nerves, and becomes a very important though much neglected factor in the diagnosis of various diseases. This group of cases exhibits a partial or total, temporary or permanent, deafness which has a tendency to recurrence, during which the functional testing may give anything from a middle ear disturbance to that of the typical lesion of the internal ear. Accompanying this may be an apoplectiform disturbance of the vestibular apparatus, as exhibited by dizziness and disturbances of equilibrium, inequalities in the after-turning nystagmus, caloric and other galvanic reactions, and interference with the kinesthetic sense. Either the cochlear or the vestibular symptoms may be uppermost, or one apparatus or the other affected separately, making the lesion as a whole overlooked or referred to other parts of the body, as is often the case when the patient is superficially examined.

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*Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Colorado Springs, Colo., March 17, 1917.

To illustrate:

Case 1.—A laborer, thirty years of age, appeared at the Stanford Clinic, complaining of an inability to navigate properly and with a ringing in one ear. He appeared to be drunk, and was examined with that in view, with a result which entirely exonerated the alcohol. His right ear was nonreactive to caloric, and he was deaf in this ear. There was a spontaneous horizontal nystagmus to the opposite side. These symptoms continued with diminishing intensity for a month while the patient was in bed, and when he left the hospital were still present to a marked degree. Three months later he returned and announced that a week following his dismissal from the hospital he had found that his liver was out of order, and had taken some China medicine with prompt secession of all symptoms. While he was in the hospital exhaustive examinations were made of the urine, feces, blood, spinal fluid, and complete physical examination by several internists, and at no time could any assignable cause be given for the neuritis, nor was there anything in his history which would point the way.

Case 2.—F. E., a laborer of thirty-five years, came to me complaining of a dizziness so great that he had been in bed for three days. He gave a history of having had three years previously a headache lasting two years, followed by a paralysis of the sixth nerve. One month before the paralysis came on the headaches became less, and ceased on recovery from the paralysis, one week later. On examination I found perfect hearing in both ears, no tinnitus; functional tests normal, save a slightly shortened bone conduction; knee jerks were all right, but the patient says they were gone one year before. He has a six-year-old child in whom I found an absence of knee jerks, but who was otherwise normal, as was also his wife. The patient exhibited a constant spontaneous horizontal nystagmus to the right, an absent caloric on the left; turning to the right produced a few jerks, turning to the left gave an extra nystagmus of fifteen seconds to the right. The next day the spontaneous nystagmus was much less, and turning in either direction gave a marked nystagmus to the right; turning to the right, a nystagmus of fifteen seconds, and to the left, a nystagmus of twenty-five seconds. There was a very slight caloric from the right, none from the left ear, so that the right vestibular nerve was now involved with the left. Hearing was perfect. Five days later the spontaneous nystagmus was gone, caloric was slow from either ear, turning always gave a nystagmus to the right of the same value as before. One and two months later the caloric was prompt from the right ear and absent from the left; the turning nystagmus was normal in direction, of small excursion and of a value of twelve to fifteen seconds. There was a negative blood Wassermann, and the urine showed an excessive amount of indican when he first came in. I placed the man on mixed treatment, but do not consider that it was at all established that there was any luetic influence in the case, and the man himself gave a very clear negative history.

Case 3.—An unmarried clerk of thirty years, a young woman under an excessive nervous strain, without a history or signs of lues, who periodically, with six or eight months' intervals, became dizzy for about a week, had to go to bed and exhibited a nystagmus to the left with an exaggeration on movements of the head, absent caloric on the right and perfect hearing.

Case 4.—An active business man of sixty years, with an arteriosclerosis, negative Wassermann, who gave a history of having previously had a slight cerebral hemorrhage and frontal headaches for twenty-five years, came complaining of unsteadiness in his walking. Functional tests gave Weber unlateralized, Rinne positive, and Schwabach slightly short; watch not heard; forks well heard; monocord at twenty-three. On turning to the right there was little or no nystagmus, but marked dizziness. On turning to the left there was a nystagmus to the right of twenty-two seconds, but the patient was not so dizzy. There was a slow caloric with small subjective signs from the left, and a prompt caloric and marked subjective signs from the right. In other words, there was an incomplete neuritis of the left vestibular nerve, due to an arteriosclerosis; and on testing this left end organ by turning there was a minimum nystagmus, but marked dizziness; and by the caloric test, both a small nystagmus and minimal subjective signs.

I have selected these cases from varied classes and ages. Probably none of them are luetic. No two exhibited similar nystagmus. None of them happen to have symptoms from the side of the cochlea, but these are well known and frequently found, so I need not burden you with them. What rôle interferences in intestinal gland secretion have in the production of these disturbances is not known, but there seems to be evidence pointing to them as the immediate cause in many cases, and the subject is under study at the present time.

I wish now to call your attention to a case exhibiting a peculiar fistula symptom, for which I have no explanation.

Case 5.—A telegrapher of thirty-five years, gave a history of having, seven years before, had a dizziness in blowing his nose, which lasted for four or five minutes, and no further disturbance until about three days ago, when he noticed the same thing. He had a marked coryza when seen; the day before, the telegraph instrument caused a sensation of pain in the right ear. There was a hissing noise in the right ear, both ear drums were slightly injected but transparent. The right drum showed some clouding at the inferior half and an atrophic area in the superior posterior quadrant. The left ear was normal; the right showed slight shrinking for the watch and whisper, seventeen and a half for monochord, fourteen and a half on the left. Weber was to the right; there was no fistula symptom. On politzerizing this patient, a marked rotary nystagmus occurred in both eyes, at first lasting a couple of minutes to the left, and then changing to the right and lasting another two or three minutes; the patient broke out in a profuse perspiration and became markedly nauseated, this lasting for an hour. The whole phenomenon was repeated three days later. Ten months later the patient heard normally in both ears, monochord at fourteen. There was no disturbance on politzerizing. I present this case without comment for your discussion.

Case 6.—There is another type of case which I wish to call briefly to your attention, and that is an acute temporary disturbance of the cochlear apparatus, due to nerve exhaustion or to acute infection in which there is an acute shrinking of the high tones and a shortened bone conduction, both of which pass off in due time, after a rest or recovery from the infection. I first noticed this in an overworked insurance agent who had had some disturbance in hearing before I saw him. He came to me complaining of an impairment of hearing, and in the course of a week I watched and demonstrated to others the rapid loss of perception for the monochord until he could no longer hear it at all with the rub, and only partially with the bow. This cleared up entirely after a couple of weeks' rest from a very nerve racking occupation.

Case 7.—The next case of this type I saw was an infection with socalled influenza (not a bacterial diagnosis) where there was a shrinking of the perception for the monochord to thirty or less, and a marked shortening of bone conduction, both of which cleared up in a week.

Rapid involvement of the labyrinth in an acute suppurative process is not frequent, but does occur, as illustrated by the following case:

Case 8.—A thirty-four-year-old office employee while in perfect health contracted a fever and became dizzy. On the following day he was told by a physician that he had tonsillitis, although the patient was not aware that there was any disturbance in his throat. On the third day the left ear drum ruptured spontaneously, and the ear continued to suppurate until seen by me, five weeks later. For the first three weeks there was an extreme dizziness and nausea with vomiting, so much so that he thought he was going to fall out of bed. The vomiting ceased after the first week. My examination showed an acute mastoiditis with a nonreactive labyrinth throughout. A Neuman labyrinth operation was done, with no dizziness immediately succeeding the operation. The organism was a capsulated streptococcus. The invasion must have taken place at the very beginning of the infection, but by what route I cannot say. He evidently had not previously had any disturbances of the ear, and no fistula could be demonstrated in the labyrinth capsule at operation. It is problematical as to whether drainage of the antrum on the second day would have saved this labyrinth, but I am inclined to believe that were I to see a case exhibiting these symptoms again, I would strongly advise such an early procedure.

Case 9.—Lastly, I wish to relate a case exhibiting the aural calamities that an individual may be subject to. A man having fallen seven floors, three years and a half ago, sustained a right suppurative ear. Two months later he was sandbagged and was dizzy for four days, so much so that he could not work. This passed off partially, the patient becoming steady by day but not at night. After the sandbagging he was stone deaf in the right (suppurative) ear, as determined by a physician. At the examination it was determined that there was considerable impairment of hearing in the other ear, the high tones being short. The caloric in this good ear was absent, there was no nystagmus on turning in either direction. The patient pointed promptly without error at the object after turning. I take it that the man had a suppurative labyrinthitis in one ear and a concussion affecting the vestibular apparatus in the other. A labyrinth operation on the right was succeeded by no dizziness. The patient exhibited the interesting phenomenon of being unable to orientate himself in space, as when diving in a tank of water.

XXIII.

ACUTE SUPPURATING MASTOIDITIS WITHOUT TYMPANITIS—PERISINUS ABSCESS, PHLEBITIS, STREPTOCOCCEMIA, OPERATION, RECOVERY.

By Charles H. Long, M. D.,

Chicago.

Acute inflammation of the mastoid process, accompanying or following suppurations of the middle ear, is a common occurrence, but is rather rare when we have an insignificant cause and no discharge from the external auditory meatus. The following case, which nearly terminated fatally, very well illustrates such cases, and reminds us of the rapid extension of certain infections without any distinct set of symptoms indicative of any one definite condition.

E. G., female, aged twelve years, student. Entered the Post-Graduate Hospital at two-thirty p. m., May 5, 1916, suffering from chills, fever, vomiting, great prostration and severe pain in the left ear.

About the middle of April she had a severe head cold; later in the month she complained of being cold and chilly, and frequently would be found sitting over the hot air register to keep herself warm. On the afternoon of May 1st, she had to leave school because of a severe pain in the left ear. The family physician, Dr. Robert Black, was summoned. Everything within the knowledge of those present was done to alleviate her suffering, but in spite of all treatment she grew gradually worse; chills, fever, retching, gagging, vomiting, sleeplessness, and delirium. This was the history submitted on her entrance to the hospital.

About four o'clock I found her exceedingly restless, with excruciating pain and tenderness over the left mastoid region. Temperature, 104.2°; pulse, 120; respirations, 36. No meatal discharge, no tympanitis, membrane red and injected. The nose and throat normal. I freely incised the ear drum, inserted a wick drain and applied external dry heat to the mastoid.

May sixth, after the paracentesis, the pain moved to the opposite side of the head and continued with great severity until I gave her ten grains of aspirin. While she had occasional pain in other parts of her body, none appeared in the head sufficiently severe to demand any special attention. The gauze was removed from the meatus, discolored with only the dried blood from the incision. The drain was not repeated.

The seventh and eighth found her free from head pain, but seemingly more toxemic, sleepless and delirious. The night nurse discovered her walking in the corridor of the hospital. There were great variations of temperature, the maximum being 104.6° at twelve o'clock noon; then she was seized with great pain in the region of the appendix and there was involuntary urination. Blood count: leucocytes, sixteen thousand.

On the ninth, after a conference with Dr. Black and the family, we decided to have Dr. Julius Grinker in consultation. On the tenth we got Dr. Grinker's report—Lateral sinus thrombosis. An X-ray was not taken because of the pain and soreness of the muscles on movement and her great prostration. Indications of meningitis were more pronounced, and there was every reason to believe a fatal termination would ensue unless an effective operation was performed.

At noon on the eleventh the simple mastoid operation was performed; the inner table was found very soft—carionecrotic. The greater portion was removed with the curette, exposing small pools of pus and masses of exudate which completely hid the sinus from view. This was inspected for fistula and examined for tension. The bone was removed in every direction until healthy dura appeared. Now that we had the vein uncovered, from the knee to the tip of the jugular bulb, I proceeded to examine the sinus with a curved applicator, pressing over the vessel to detect a thrombus, but it seemed to be free from coagula. However, to make certain, I made a long incision in the sinus. The blood gushed freely from both ends —no clots appearing, the hemorrhage was controlled by packing. Satisfied with the completeness of the operation, a moist dressing was applied and the patient sent to her room.

May twelfth, while no marked improvement had occurred, she was certainly no worse. The tongue was moist and the meningeal irritation had not increased. The laboratory reported streptococci in the pus.

May thirteenth, there being so little change in her condition, I made a spinal puncture, and found the spinal fluid normal and not under pressure. From then on I was inclined to adopt the "watchful waiting" method of treatment, which in this particular case seems to have met with success. As her blood was freed from the poisonous and septic bacteria, her system became normal and her organs performed their functions in a regular and orderly manner. She was able to leave the hospital June 3d—twenty-three days after the operation, or thirty-four days from the date of leaving school.

The following points in the case are worthy of especial consideration:

First, the mildness of the primary infection.

Second, the transmission of such virulent bacteria from the nose to the mastoid cells without injury to any of the structure during the journey.

Third, the six days of severe pain and tenderness over the mastoid region, then an abrupt cessation.

Fourth, the extensive necrosis of the mastoid found at operation.

Fifth, the fact that no thrombus was found in the sinus.

Sixth, the uselessness of nasal douches and sprays as a prophylaxis of ear infection.

Seventh, the tardiness in operating.

Eighth, the slow convalescence.

A common cold in the head so seldom sets up a mastoiditis without first attacking the middle ear, perforating the drum membrane, and delivering a secretion from the meatus, that I feel we are justified in deferring operative procedures until we have decided upon its necessity and the probability of its success.

It is now quite clear that about May 1st this infection had located itself within the mastoid cells. These cellular walls, being bony, withstood the strain of the violent septic process for six days, when suddenly they weakened and the contents made their way in the direction of least resistance, which was the dura. The pain and tenderness subsided when the pressure was removed, but the septic and meningeal symptoms continued with increased severity. There was good reason to suspect a sinus thrombosis, with or without extradural abscess, or a meningitis, because we had the classical symptoms of sinus thrombosis—viz., chills and fever with its characteristic marked remitting course, as indicated by the temperature chart; and accompanying these we had the nausea, vomiting, delirium, decided prostration, rapid pulse and respiration, stiffness of the neck, optic neuritis, all of which were very significant of intracranial complications; yet we had not been able to demonstrate the existence of pus in the mastoid by any well defined local manifestations.

In 1907 I witnessed the late Dr. Gruening of New York operate on a young woman manifesting these classical sinus thrombosis symptoms; but the operation, though exposing the sinus very plainly, failed to demonstrate any septic process. That case had made its impression upon my mind, and I was inclined to wait as long as it was safe.

Although Dr. Black gave me entire control of the case, I was glad—for two reasons—of his continued interest: First, and most important, his counsel. Second, to learn the result of the nose and throat cleansing solutions which he had prescribed from the beginning and which I had not opposed. I do not think that many specialists are using them today, because they have learned that these solutions are liable to do more harm than good; and as a prophylactic to ear infections they are worse than useless. They are a relic of the past. As the general practitioner continues to use what he reads, he should be cautioned of their evil effects on the middle ear.

XXIV.

THE PRACTICAL USES OF RECENT WORK ON THE INTERNAL EAR, TO THE GENERAL PRACTITIONER, THE OTOLOGIST, OPHTHALMOLOGIST, SYPHILOLOGIST, NEUROLOGIST AND SURGEON.*

By Isaac H. Jones, A. M., M. D.,

Philadelphia.

Any work in physiology may be regarded as an "interesting study," but from the standpoint of most of us in the practice of medicine, research work of any kind becomes of greatest importance when it proves useful in clinical work. The cry is: "That is all very interesting, but of what use is it? How does it help sick people?" To answer this question is the spirit of this paper. A study of the relation of the ear to the central nervous system not only opens a new field for investigation in physiology, but also furnishes information of diagnostic value in the every-day examination of patients. Although it is essentially and entirely an ear study, and is useful in the analysis of ear conditions, we wish to emphasize that recent investigations have shown it to be of value in medical and surgical diagnosis.

It is now recognized that the internal ear, instead of being one organ, consists of two organs of different functions—the cochlea, which is the organ of hearing, and the static labyrinth, which is the organ of equilibration. It seems very remarkable that whereas the cochlea has long been recognized as the organ of hearing, the static portion of the ear, consisting of the utricle, saccule and three semicircular canals, has been through all the years a "terra incognita." It is only in the past few years that the function of the static labyrinth has been carefully studied, preeminently by the Vienna group of otologists, to whom we are indebted for the new methods of testing the internal ear. Robert Bárány received the Nobel prize of 1915 for this work, and he is to be regarded as the pioneer in the clinical application of the study of the ear and the central nervous system.

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THE TESTS.

The semicircular canals may be stimulated by revolving a person in a turning chair, or by douching the ears with either cold or hot water, or by applying the galvanic current to the ear. According to our experience, the turning and douching are much more useful than the electric test. The galvanic current stimulates equally all portions of the internal ear, and in fact the eighth nerve itself. As we regard it of greatest importance to analyze the function of the canals separately, we will consider in this paper only the tests by turning and douching. The essential feature in the physiology of these tests is that the endolymph within the particular canal or canals stimulated is set in motion on the one hand mechanically by the turning, and on the other hand thermically by the douching. When an individual is turned to the right, the endolymph catches up to the movement of the body, and when the turning chair is stopped, the endolymph continues to move to the right by its own momentum. The physiology of the caloric test may be illustrated by the "thermosyphon" system of some of the popular automobiles. They have no water pump to force a circulation through the radiator; the engine heats the water, decreasing its specific gravity, and causing it to move upward and forward into the radiator, where it is cooled, drops to the bottom of the radiator and continues downward and backward into the engine, thus completing the circuit. Similarly chilling the outer portion of a semicircular canal increases the density of the endolymph at this point and produces a circulation downward.

Such ear stimulation produces certain definite phenomena in normal individuals—a rhythmic jerking of the eyes, known as nystagmus, and a subjective sensation of turning, which may be termed a systematized vertigo. The eyes are always drawn in the direction of the endolymph movement, and are then quickly jerked back in the opposite direction by impulses from the cerebrum. The subjective sensation of vertigo is always in a direction opposite to the endolymph movement. According to our conviction, there are only two reactions produced by ear stimulation—vertigo and nystagmus; it is because of this vertigo that the individual falls in a definite direction, and also when he attempts to find with his finger an object he has previously touched, he is unable to find it, but "past-points" to the right or left, above or below, depending on the direction and plane of his sensation of vertigo.

It is evident that if stimulation of the ear causes a movement of the eyes, there must be a definite nerve pathway from the ear to the eye muscles. Also if ear stimulation produces dizziness, it is evident there must be a nerve pathway from the ear to the cerebral cortex. The special object of our work has been the attempt to determine the pathways through the brain from the ear. On the basis of over three hundred and fifty pathologic cases, including a considerable number of operations and autopsies, we have come to certain conclusions as to the course of the fibers from the ear through the brain stem, cerebellum and cerebrum. In brief, our conclusions are as follows: The fibers from the horizontal semicircular canals pass through the eighth nerve to the brain stem, and enter Deiters' nucleus in the medulla oblongata. At this nucleus the fibers divide, going on the one hand to the posterior longitudinal bundle, through which they are connected with the various eye muscle nuclei, to be distributed through the third, fourth and sixth nerves to the eye muscles themselves. It is this pathway that is responsible for the eye movement. The other pathway goes from Deiters' nucleus through the inferior cerebellar peduncle to the cerebellar nuclei of the same side, from which it proceeds through the right superior cerebellar peduncle to the decussation of the two superior cerebellar peduncles in the base of the cerebral crura; from this point there are two pathways to the cerebral cortex of both sides, but the main pathway goes to the cortex of the opposite side. The cortical centers that receive these fibers are postulated by Mills to be in the posterior portion of the second temporal convolutions adjacent to the cortical areas for hearing. It is this pathway from the horizontal canal to the cerebral cortex, passing through the cerebellum, that is responsible for the production of vertigo on ear stimulation. The fibers from the vertical semicircular canals have a different pathway after entering the brain stem, ascending in the pons to a point above the middle of the pons. At this point the fibers divide, going on the one hand to the posterior longitudinal bundle, to be distributed to the eye muscles; on the other hand the fibers enter the cerebellar nuclei through the middle cerebellar peduncle, from which point their pathway is the same as that of the fibers from the horizontal canal. In a nutshell, by the vestibular apparatus we mean the static portions of the internal ears and the pathways from the ears through the brain stem, cerebellum and cerebrum.

IMPORTANCE OF THESE TESTS IN DIAGNOSIS.

1. The general practitioner is constantly confronted with cases of "vertigo." It is surely not overstating the fact to say that at best he can only guess as to its significance. In the light of these new tests, vertigo should no longer be regarded as a vague and general symptom. We should never speak of gastric vertigo, kidney vertigo, cardiovascular vertigo, idiopathic vertigo, or any other generalization which in its ultimate analysis means nothing. Vertigo is definitely a disturbance of the equilibratory apparatus. These tests give us the means of analyzing this apparatus. All conscious sensations are cerebral; headache, from whatever cause, is a disturbance in the brain. Similarly, vertigo is due to an irritation, impairment or destruction of some portion or portions of the ear or its associated pathways. Therefore, just as we examine the urine in suspected cases of nephritis or diabetes, or as we have a Wassermann test made in suspected cases of syphilis, just so these ear tests enable us to analyze the apparatus responsible for dizziness. Instead of guessing, we then know the reason for the vertigo. We do not hesitate to say that it is very rare that a "dizzy" case remains obscure after these ear tests, and in most instances the diagnosis becomes clear and simple. After these tests are made we are able to say, for example, that the patient is dizzy because of toxic impairment of his labyrinth, a labyrinthitis, an organic lesion in some

part of the vestibular tract such as is produced, for example, by a brain tumor, internal hydrocephalus or multiple sclerosis; or in the presence of normal reactions, we can state with assurance that the vertigo is purely functional.

2. Value to the otologist. It is very surprising how much information can be obtained by these tests in the ordinary everyday study of an ear case. Briefly, from the ear standpoint alone, we have by means of the turning and douching tests, produced nystagmus, vertigo, past-pointing and falling, the clean-cut quantitative objective evidence of the function of each part of the static portion of the labyrinth and eighth nerve. A patient complains of deafness; the question arises, is it a middle ear or an internal ear condition? These hard and fast reactions give positive data of the function of the internal ear; then a positive opinion can be given with less reservation. Deaf and dumb institutions obviously should have a turning chair; in these poor unfortunates the functional tests of hearing are so often unsatisfactory; we never feel after making the hearing tests that we are sure the labyrinth is completely destroyed. It is just here that the new tests are valuable; they either do or do not give reactions. The answer is "Yes" or "No." In routine examination, it is, of course, not necessary to conduct all the tests; merely by turning the patient and obtaining twenty-six seconds of after-turning nystagmus, we at once can conclude that both static labyrinths are normal. Very often, however, the nystagmus will be subnormal, and immediately we have a hint as to the necessity for further study. We have no hesitancy in suggesting that if the otologist in his routine work will make such a simple test, he will be surprised to find that in many instances an entirely new light is thrown upon the condition of his patient.

3. Value to the ophthalmologist. The attention of the eye specialist has not been called to the intimate relation between the ear and the eye. He should surely know that a nystagmus of any type and in any direction can be produced "to order" by ear stimulation. There is an absolute and definite relation between the canal stimulated and the type of nystagmus produced. If we wish to produce a vertical nystagmus upward, we place the patient's head over toward the right shoulder, turn him to the left, and there appears a vertical nystagmus up-

ward. If we wish a rotary nystagmus to the left, we douche the right ear with head upright with cold water, or douche the left ear with hot water, and there is produced a rotary nystagmus to the left. Although space does not permit discussing the reasons for this marvelously accurate ear-to-eye mechanism, yet the above gives some idea of the fixed relationship between the semicircular canals and the eye muscles. The "fistula" test serves as an illustration; if there is caries of bone in the outer wall of the labyrinth producing a fistula into the internal ear, or, for that matter, if the stapes is unduly mobile in the oval window, applying a Politzer bag into the ear and causing pressure or suction, causes the eyes to dance violently. Recently a well known ophthalmologist seeing us do this test expressed his astonishment—"I had no idea there was such close relation between the ear and the eye."

THE PRACTICAL APPLICATION OF THESE TESTS.

(a) In the study of eye muscle palsy. For example, loss of conjugate deviation in various directions should always be studied by means of the ear tests. There is a loss of voluntary control; the patient is unable, when asked, to look in a given direction. If the ear stimulus causes the eyes to move in this direction, it shows without question that there are normal pathways from the ear through the eye muscle nuclei and through the nerves to the eye muscles themselves—therefore, the lesion is supranuclear.

(b) Spontaneous nystagmus may be ocular in origin from corneal scars, macular lesions, or eye strain as in miners, in which case the cause is easily determined by proper ocular examination. But what of those cases in which the lesion lies not so near the surface? It is in such cases that the ear tests are essential. Recently there were two babies, both showing a wild oscillatory nystagmus. Naturally it was the ophthalmologist who was called on to determine its cause; he found nothing wrong in the eyes themselves to account for the nystagmus. The ear tests, however, showed a striking difference between the two cases. On one we reported: "The ear tests suggest an irritation of the vestibuloocular tract, but no organic impairment; the prognosis is good." The child had a perfect recovery. The report on the other patient was:

"There is an organic block of the vestibuloocular tracts of both sides in the brain stem; the prognosis is bad." This patient died. The reason for this report was that the first patient on ear stimulation immediately showed a change of the oscillatory nystagmus into a definite rhythmic pull. The ear stimulation, as it were, seized both eyes and caused them to move rhythmically in the proper direction; evidently, therefore, there was no organic block of the pathways. The other case continued to have the oscillatory nystagmus and was entirely unaffected by ear stimulation, showing a definite organic block of the pathways from both ears to the eyes.

4. The ear and the syphilologist. Syphilis is such a prevalent affection that any method of diagnosis deserves thorough investigation. The disease manifests itself in so many forms that although it is comparatively easy to diagnose in certain cases, it is extremely difficult in others. There is probably no other disease where early diagnosis is so important; if detected early, certain well known remedies can often effect a prompt and permanent cure. Prognosis, in fact, depends largely upon how quickly the treatment is instituted. Discovery of the Wassermann test was justly hailed as an extraordinary aid to the clinician, but unfortunately the Wassermann reaction is not absolute; it is common experience to find cases that are clinically undoubtedly syphilitic, and which also improve under antisyphilitic treatment, but which are repeatedly negative to the Wassermann test. Our investigations to date have led us to believe:

(a) That the new ear tests may prove useful in the early diagnosis of syphilis. Many cases referred to us show a beginning very slight but unmistakable impairment of eighth nerve function only a few weeks after the primary infection; had it not been for the delicacy of the new tests, we would in all probability have not been able to detect this impairment for months.

(b) Ear examinations unquestionably are valuable in detecting a beginning involvement of the central nervous system. It is recognized that the nervous system is attacked in five per cent of those infected; we venture to suggest that probably this percentage is much higher, and that many cases are not recognized because there is no obvious involvement, as in locomotor ataxia, facial paralysis or general paresis. The eighth nerve is particularly vulnerable to this disease, and analysis of the function of the eighth nerve and the intracranial pathways from the eighth nerve may in certain cases demonstrate a beginning involvement of the nervous system several years before it can be detected by any other method.

5. Value to the neurologist. Naturally all the subjects already mentioned are of interest to the neurologist; the determining of the cause of vertigo, the degree of function of the eighth nerve and labyrinth, the study of eye muscle palsies, the analysis of spontaneous nystagmus, and the early diagnosis of syphilis of the central nervous system. In addition, the neurologist has in these tests a very definite help in:

(a) Differential diagnosis between labyrinth and intracranial lesions. The ear tests are indispensable for such differentiation. In so many instances the symptoms of internal ear disturbances and of cerebellar lesions are identical. To illustrate: A patient was given a neurologic diagnosis of lesion of the right cerebellar hemisphere; ear tests gave additional data to the neurologist and demonstrated conclusively that it was a lesion of the right labyrinth.

(b) Intracranial localization. The accuracy of the ear tests has been very surprising in locating lesions in the cerebellar pontine angle, medulla oblongata, pons, cerebellar peduncles, cerebellum and various portions of the cerebrum, including the parietal lobe, temporal lobe and occipital lobe. Neurologists invariably want an eye examination in intracranial cases. We as otologists are not in a position to state the relative values, but the neurologists and ophthalmologists most acquainted with these ear tests assert that of the two methods of approach, very much more definite information can be had from the ear tests than from the eye examination.

6. Value to the surgeon. Perhaps no phase of surgery is so difficult in diagnosis as intracranial localization. It is a sad commentary on intracranial surgery that in a large majority of cases the surgeon must content himself merely with a palliative decompression. The ear tests have proven of real value in the locating of lesions; but in addition they may prove helpful to the surgeon in preventing unnecessary operations. This may be illustrated by the following two cases: A woman appeared to have a tumor of the right cerebellar hemisphere.

This diagnosis was confirmed by neurologic consultants, and the X-ray report stated that it was a cyst in the right cerebellar hemisphere. The surgeon was about to operate, but hesitated because the ear report said it was a lesion of the right labyrinth, and that the cerebellum appeared intact. The woman had a perfect recovery without operation, has had a baby, and has remained in perfect health ever since—two years. Another woman was operated on for carcinoma of the gall bladder; the findings were negative. Subsequent ear examination made it perfectly simple that all her symptoms—nausea, vomiting, diarrhea and consequent emaciation—had been due entirely to recurring attacks of labyrinthitis. She made an excellent recovery. In the opinion of the surgeons most acquainted with this work, no brain should be opened without giving the patient the benefit of the ear tests.

In conclusion, it must be remembered that the work of the ear specialist along the lines of the relation of the ear to medical and surgical diagnosis is only in its infancy. This paper is intended not so much to call attention to what we have done, as to hint at the future possibilities of this study and to indicate how the cooperation of the otologists with those in other branches of medical work can "help sick people."

XXV.

OBSERVATIONS ON AFTER-TURNING NYSTAGMUS.

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I am prompted to present a paper on the subject of after-turning nystagmus for the reason that my investigations, covering a period of eight years (1906 to 1914), have resulted in findings contradictory to those of Bárány, which have been accepted without challenge and as final by the otologic profession. I believe the observations of Bárány on after-turning nystagmus have been faulty because of inaccurate technic, and I trust to prove this contention in the course of the present paper. Furthermore, it is my intention to show that the wide discrepancies in the duration of after-turning nystagmus in normal individuals, claimed by Bárány, do not exist. It is because of these marked discrepancies that certain authorities are inclined to accept the labyrinthine tests with some doubt. It is my belief that a review of my findings, which can be readily corroborated by anyone willing to investigate, will tend to dispel the doubt and permit us to accept the turning test as a valuable one in the diagnosis of labyrinthine conditions.

While my intention is to point out certain errors made by Bárány, there is no intention to reflect upon his otherwise excellent service in the field of otology.

Purkinje, in 1820, was perhaps the first to make observations of vertigo and nystagmus on humans by turning. He, however, did not associate the symptoms with the semicircular canals, but with the brain.

It was left to Flourens, in 1842, to discover the law that each semicircular canal produces eye movements in the plane of the canal, and that when more than one canal was irritated simultaneously, eye movements occurred corresponding to the resultant of the canals irritated. He conducted experiments upon animals (pigeons and rabbits), sectioning the semicircular canals, from which he concluded that the canals are the seat of an organ which normally had a controlling influence upon body movements. In 1861 the same author experimented upon animals, extirpating the cerebral hemispheres, when he found the semicircular canals reacted after the same manner as before, from which he justly concluded that the reactions were entirely independent of the great brain (cerebrum).

Chevreul, in 1831, made experiments much after the same manner as Flourens; he differed, however, in his conclusions. While Flourens believed the pendulum-like motions and deviations of the head and neck from sectioning of the canals were those of irritation, Chevreul believed them to be manifestations of loss of function from destruction. Since then turning experiments have been made by Crum-Brown, Breuer, Mach, Delage, Kreidl, Kubo and to less extent by others.

It remained for Bárány, however, to conduct the more extensive turning experiments on humans, both normal and pathologic, and to assign to the experiments their place as tests in the diagnosis of labyrinthine conditions. Notwithstanding Bárány's painstaking experiments, he has overlooked certain precautions which have led him into uncertainties which might have been avoided had he observed these precautions.

Bárány, on page 18 of his book, "Physiologie und Pathologie des Bogengang-Apparates beim Menschen," recommends the use of opaque spectacles advised by Hans Abels in timing horizontal after-nystagmus. In the following paragraph he tells why it is advisable to have the subject wear the opaque spectacles, viz., to exclude the physiologic nystagmus that results from the subject fixing his eyes in a side position. In the next paragraph, in referring to rotatory after-nystagmus, he discards the opaque spectacles as unnecessary and uses his fixation apparatus, which consists of a head band to which is fastened a horizontal bar extending forward from the forehead plate about fifteen centimeters. At the distal end of this horizontal bar is attached a vertical bar about four centimeters long; the lower end terminates in a small sphere at which the subject is directed to fix his gaze. He then begins the citation of his findings.

At the bottom of page 18 and the top of page 19 he claims to have examined two hundred normal individuals for horizontal after-turning nystagmus after ten turnings to the right, and found the average duration forty-one seconds, while the after-nystagmus to the left averaged thirty-nine seconds. He also records one individual who showed no after-turning nystagmus after ten turns. The maximum was for nystagmus to the right ninety-eight seconds, to the left one hundred and twenty.

On page 19 he says that we see both extremes—the case with hardly any after-nystagmus and the case with two minutes' duration. Under physiologic conditions there exists great differences in the duration of after-turning nystagmus. He claims that it is not infrequent to find the duration of after-nystagmus in one case to be about three or four times as long as that in another.

In sixty individuals examined repeatedly on the same or on different days, he observed after-nystagmus averaging forty seconds; maximum eighty seconds, minimum twenty-five seconds. He found that only one-fifth of the cases with after-turning nystagmus exceeding forty seconds showed a constancy in the duration of the nystagmus; while the difference ranged from ten to thirty-eight seconds. In other cases with an average duration of after-turning nystagmus of less than forty seconds, the variation ascertained at different examinations ranged from four seconds to twenty-three seconds. He rules out fatigue as a possible explanation, since the examinations were made at times sufficiently far apart to allow for ample rest between.

On page 21, second paragraph, Bárány claims that in a number of cases in which he tried both rapid and slow turnings, he found that after ten rapid turns the nystagmus lasted from eighteen to nineteen seconds, and after slow turns from twenty-six to thirty seconds. He qualifies this statement in a subsequent sentence by saying that in half the cases he found the rapid turns produced a longer after-turning nystagmus than slower turns. Lower down on the same page he claims that with a great number of persons the horizontal nystagmus after twenty, thirty, forty, fifty and sixty turns was the same as after ten turns, adding that "it appears that in the average, ten turns show the highest worth, while with a greater or less number of turns the amount (duration) diminishes." He claims that the amount (duration) of after-turning nystagmus after twenty turns is about equal to that after five turns. He grants, however, that the nystagmus is more pronounced (intensive) after twenty than after five turns. According to Bárány, after twenty or more turns there appears not infrequently a nach-nach-nystagmus (after-after-nystagmus), which begins a few seconds after the cessation of a very intensive after-nystagmus and lasts sometimes a minute.

Concerning dancers, Bárány claims an interesting finding, namely, that in those who habitually turn to the right, in Austria referred to as rechtstänzer, the duration of the horizontal turning nystagmus to the left (thirty seconds) falls considerably shorter than that to the right (forty-nine seconds). Furthermore, he has never seen the after-nystagmus, in such cases, to the left to last longer than that to the right.

On page 25 Bárány confesses that he is unable to explain why at one time the after-nystagmus to the left exceeds that to the right, while at another time the nystagmus to the right exceeds that to the left. He confesses, furthermore, that he is unable to explain why the nystagmus after twenty turns should be less than after ten turns; and, too, why there should occur an after-after-nystagmus, of one minute's duration, to the opposite side following the after-nystagmus. In his attempts to explain these incongruities he frames up a theory which places the blame upon two vestibular centers in Deiter's nucleus.

Since 1907, when Bárány first presented his findings on after-turning nystagmus, authorities generally have accepted his figures as conclusive, on faith, and incorporated them in their general works on otology as well as in their special works on the labyrinth, including those written by Ruttin and by Braun and Friesner. Others, somewhat more independent, have accepted Bárány's findings, but have ventured their own explanations as substitutes for those of Bárány.

Since Bárány is the one who deserves the credit for having made the most extensive experiments with after-turning nystagmus, and is the most recognized and most quoted authority on the subject, I feel justified in paying him the honor by directing my controversy towards him, in the hope of convincing him and the otologic profession that his findings are incorrect. I wish to call your attention further to the work of Bárány on after-turning nystagmus by citing from a paper presented by him at the Ninth International Otological Congress,* Boston, August, 1912, and subsequently published in the proceedings under the title of "The Relationship Between the Semicircular Canals and the Eye Muscles, the Central Mechanism in Vestibular Nystagmus." In referring to horizontal after-nystagmus he says: "Stabartz Hofer, at my request, investigated a number of normal cases with no spontaneous nystagmus present, to determine the position in which an exactly horizontal nystagmus would be present in every direction of the visual axis. I controlled his findings. We observed that a pure horizontal nystagmus was never present in a position described by Bruning, but that if turning to the right side is considered, there is always horizontal or rotatory nystagmus to the left when looking to the left." * * * "In another series of experiments I have observed that if with a normal person you produce a strong rotatory nystagmus by turning him with the head ninety degrees forward or backward, you will notice that in every direction of vision the nystagmus will be nearly purely rotatory."

In the same proceedings, in a paper on the "Differential Diagnosis Between Labyrinth Suppuration, Serous Labyrinthitis," etc., page 275, under the heading "Remarks 1," he says: "Spontaneous nystagmus is examined by letting the patient look at the doctor's forefinger, held at a distance of three-fourths of a meter."

In his table for differential diagnosis of the mentioned diseases, in the column under suppuration of the right labyrinth since twenty days, also in that after six months, and under serous labyrinthitis, he mentions that the patient has spontaneous nystagmus to the right as well as to the left side.

I have cited Bárány quite fully, in order that we do him no injustice. I shall now list my table of cases with the findings, after which I shall summarize them and contrast them with Bárány's. Finally, I shall attempt an explanation of the reasons for the disparity that exists between his and my own findings.

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*The paper was presented but not read, owing to his inability to be present at the Congress.

I have listed a group of normal cases, a group of cases with middle ear conditions, but with normal internal ears, a group with one-sided internal ear destruction, a series of deafmutes with partial and others with complete destruction of both ears, and a number of others (odd cases) that cannot be classed in any of the above groups. A greater number of cases might have been entered in our list, particularly normals, but the records were found to be too incomplete for the purpose. These occurred in patients complaining of symptoms which vaguely suggested the possibility of internal ear trouble, but upon examination were found to be normal from this standpoint. The results in these cases compared with those in our list. More tests were made for horizontal than for rotatory after-nystagmus, for the reason that the patients tolerated the rotatory less. There were many tests made for rotatory after-turning nystagmus upon charity patients, to whom I offered money for the privilege, but after a few attempts they balked, in spite of the offer, so that complete examination in many cases could not be made. I had to omit them from the list. The results, as far as they went, were in accord with those in the list.

The List of Cases.

Among the twenty-four normal cases, and the eleven cases of middle ear trouble with no spontaneous nystagmus or history of labyrinthine involvement, the longest duration of nystagmus to the right after five turns to the left was nineteen seconds in Case 5. The longest duration of nystagmus to the left after five turns to the right was nineteen seconds in Case 13. The shortest duration of nystagmus to the right after five turns to the left was four seconds in Case 18. The shortest duration of nystagmus to the left after five turns to the right was four seconds in Case 18. The average duration of nystagmus to the right after five turns to the left was a trifle under thirteen seconds, while the average duration of nystagmus to the left after five turns to the right was a trifle over thirteen seconds.

The longest duration of nystagmus to the right after ten turns to the left was twenty-nine seconds in Case 22. The longest duration of nystagmus to the left after ten turns to the right was thirty-one seconds in Case 2. The shortest duration of nystagmus to the right after ten turns to the left was ten seconds in Case 10. Since this case was that of a whirling dervisher and a left turner, we may omit his case for the present and consider the next shortest, that of fifteen seconds in Case 17. The shortest duration of nystagmus to the left after ten turns to the right was sixteen seconds in Case 17. The average duration of nystagmus to the right after ten turns to the left was a fraction of a second less than twenty-four seconds. The average duration of nystagmus to the left after ten turns to the right was twenty-four seconds. The reduction in after-nystagmus to the right in Case 10 (whirling dervisher) brought the figures for the average the slightest fraction under twenty-four seconds. If this case was excluded the duration would equal about twenty-four seconds.

The longest duration of nystagmus to the right after fifteen turns to the left was thirty-two seconds in Cases 7, 20 and 22. The longest duration of nystagmus to the left after fifteen turns to the right was thirty-two seconds in Case 22. The shortest duration of nystagmus to the right after fifteen turns to the left was eleven seconds in Case 10. Excluding this one we have that of seventeen seconds in Case 11. The shortest duration of nystagmus to the left after fifteen turns to the right was twenty seconds in Cases 10 and 11. The average duration of nystagmus to the right after fifteen turns to the left was a trifle over twenty-six seconds, while the duration of nystagmus to the left after fifteen turns to the right was a trifle over twenty-six and one-half seconds.

The longest duration of nystagmus to the right after twenty turns to the left was thirty-six seconds in Case 22. The longest duration of nystagmus to the left after twenty turns to the right was thirty-six seconds in Case 22. The shortest duration of nystagmus to the right after twenty turns to the left was seventeen seconds in Case 10, next to this it was twenty-three seconds in Case 17. The shortest duration of nystagmus to the left after twenty turns to the right was twenty seconds in Case 15. The average duration of nystagmus to the right after twenty turns to the left was a trifle over twenty-eight seconds. The average duration of nystagmus to the left after twenty turns to the right was about twenty-eight seconds. In no instance where multiple turns were tried did after-nystagmus to either side after five turns exceed that after ten turns. Nor did the after-nystagmus after ten turns exceed that after fifteen turns, nor did the after-nystagmus after fifteen turns exceed that after twenty. There were cases, however, where the figures after a greater number of turns did not exceed those after a fewer number of turns, but these were exceptional.

The widest discrepancies in the duration of the after-turning nystagmus occurred after five turns—the shortest four seconds, the longest nineteen seconds. The next widest discrepancies in duration occurred after ten turns—the shortest, excluding Case 10, was fifteen seconds, the longest thirty-one seconds. The next occurred after fifteen turns, when the shortest duration was seventeen seconds and the longest thirty-two seconds. The least discrepancy occurred after twenty turns, when the shortest, excluding Case 10, was twenty-three seconds and the longest thirty-six. This would indicate that a twenty turn test is more reliable than a fifteen turn test, that a fifteen turn test is more reliable than a ten turn, and that a ten turn test is more reliable than a five.

Above we have referred only to horizontal nystagmus. Concerning rotatory nystagmus, too few cases have been collected from which to form any conclusion. However, from these few we note an average of twelve seconds to each side after five turns; eighteen seconds after ten turns; excluding Case 10, the average is twenty seconds. We note, too, a greater duration after ten turns than after five, and an increase after fifteen turns over that after ten.

In the list there are ten cases of one-sided labyrinth destruction, eight cases complicating chronic middle ear suppuration and two complicating acute middle ear suppuration. Only two cases, Nos. 45 and 47, were tried with multiple turnings. In Case 45 the after-turning nystagmus after fifteen turns fell below the figures after ten, and this was so on both sides. The other case, 47, showed a consistent increase excepting for the rotatory after-nystagmus to the left, when it was found that the duration after fifteen turns equaled that after ten. All ten cases were turned at least ten times, either with head erect or bent forward ninety degrees, and some were turned after both methods. Of six who were turned for horizontal nystagmus, the longest duration to the unaffected side was thirty-five seconds in Case 39, the shortest ten seconds in Case 40. The longest duration to the affected side was sixteen seconds in Case 45, the shortest five seconds in Case 40. The average to the unaffected side was twenty-four seconds, to the affected side a trifle under twelve seconds. Eight were turned for rotatory nystagmus. After ten turns the longest duration to the unaffected side was twenty-three seconds in Case 41, while the shortest was ten seconds in Case 40. The longest duration to the affected side was thirteen seconds in Case 37, the shortest was five seconds in Case 40. The average duration to the unaffected side was sixteen seconds, to the affected side eight seconds.

There were four cases of syphilis involving the inner ear or nerve; each differed somewhat from the other, so that we are compelled to consider them separately.

In Case 46 there was partial reduction of hearing in both ears, worse in the left, with Rinné positive on both sides. There was relative reduction to turning and galvanism, particularly on the left side. The case was seen during the stage of improvement which followed antisyphilitic treatment.

In Case 44 there was syphilitic involvement of both sides; on the right the hearing apparatus was more involved than on the left, and the vestibular apparatus about equally on both. The after-turning nystagmus to the two sides was markedly reduced after ten turns—i. e., thirteen seconds to the right and twelve seconds to the left.

In Case 48 we have a typical one-sided destruction (right) of both hearing and vestibular apparatus. After ten turns the horizontal nystagmus to the right was seven seconds, to the left fifteen seconds. The other findings, fourteen and twenty seconds, and those for rotatory nystagmus of thirteen and fourteen seconds, were ascertained late in the case—after the patient had improved somewhat. The findings seven and fifteen seconds were at the height of the affection. This patient subsequently improved considerably with the administration of iodid of potassium internally. I might say, in passing, that we have found recent cases of syphilitic ear manifestations quite amenable to treatment.

In Case 49 we have a late syphilitic affection of both ears, with marked reduction of hearing and marked reduction of the vestibular function, as shown by the turning test. This patient was turned on several occasions and presented consistently the same findings. After ten turns the horizontal after-nystagmus was twelve seconds to each side. The figures after five turns about equal those after ten turns; after fifteen turns the figures increased slightly; after twenty turns they increased still more than after fifteen turns. In this respect—that is, increasing after fifteen and after twenty turns—she behaved like the normal cases.

Thirty-two deafmutes were examined and are here listed, among which nine were attributed to meningitis. All but one of the meningitis cases were completely deaf on both sides. Case 58 had slight remains of hearing on both sides. All but one case, 54, showed absence of reaction to turning. That is, after ten turns the patients manifested no nystagmus or dizziness. Ten cases of deafmutes were of congenital origin; Cases 61 to 70, inclusive. Of this number three had no reaction from vestibular apparatus; they were Cases 61, 64 and 70. Seven cases showed complete bilateral deafness, and three escaped with slight remains of hearing. Those escaping were Cases 62, 67 and 70. The comparison of these findings shows that the cochlear apparatus is more vulnerable than the vestibular, and in our group may be represented by the proportion of seven to three. In our list of deafmutes we have five cases, from 74 to 78, inclusive, where the cause is assigned to head injuries. One case, 78, shows complete deafness on both sides. Three cases, 74, 76 and 77, have slight remains of hearing, and one case, 75, shows complete deafness on the left side and slight remains on the right. Two cases, 74 and 78, had no reaction to turning. The others showed positive but reduced reaction to both sides. Two cases, 71 and 72, were due to measles. Both were completely deaf on both sides. Case 71 presented no reaction to turning, and Case 72 presented positive reaction to turning, but with reduced figures. One case, 79, was deaf following scarlet fever. The child was completely deaf on both sides, with no reaction to turning. One case, 80, was completely deaf in both ears following typhoid. This case manifested no reaction to turning. One case, 82, attributed to whooping cough, was completely deaf in the right ear, but had slight remains in the left. The patient presented no reaction to turning. Three cases, 52, 73 and 81, were deaf from unknown causes. All three cases showed slight remains of hearing in both ears. Cases 52 and 73 showed positive but reduced reaction to turning. Case 81 showed no reaction to turning.

Case 83 was an odd one. The patient claims that his deafness came after a cerebral hemorrhage. If he is right in this we must conclude that the deafness was of central origin, the lesion being on the left side. His vestibular function escaped, for he shows a relatively normal after-turning nystagmus to the two sides.

Case 85 deserves special mention. She was a girl, seventeen years of age, who had developed acute mastoiditis following the syringing of the right ear for the removal of wax, but not at my hands. When seen by the writer the patient was suffering from pain, fever, marked tenderness over the ear and mastoid. At the time of operation the prominence of the horizontal semicircular canal showed a fistula. This was anticipated before operation, on account of the already existing horizontal nystagmus to the left becoming decidedly more pronounced by finger pressure in the left ear. It is impossible to include everything in the table, but the end results were remarkable in that she retained function in the vertical canals, so that the case may be classed as one of circumscribed labyrinthitis.

In our list are nine cases of bilateral middle ear suppuration. Cases 84, 93, 94, 95, 96, 97, 101, 106 and 117. Case 94 showed healing on the left side. The hearing was more or less reduced in all nine cases on both sides. Four cases, 95, 97, 106 and 117, showed undeniable reduction of after-nystagmus to the two sides. Case 97 was tried after multiple turns for both horizontal and rotatory nystagmus. Two cases, 84 and 96, showed relatively normal reactions to turnings; the rest showed more or less reduction to one or both sides. Except in Case 84 none showed figures above the average, and this case did not exceed the high marks of the normals. Where multiple turns were tried, we find again an increase in the duration of the nystagmus with an increase of the number of turns up to twenty. There were occasional differences on the two sides when we compared the horizontal with the rotatory, but this could readily be expected in cases nearing a circumscribed labyrinthitis.

There were four cases of chronic middle ear suppuration. Cases 98, 99, 100 and 113. Case 99 showed marked deafness of the perceiving apparatus type. This, taken with negative reaction to cold water and with reduction of the after-nystagmus particularly toward the affected side, forced me to the conclusion that the inner ear of that side was completely destroyed. The after-nystagmus to the right, after ten turns, was six seconds, and to the unaffected side twelve seconds. This is the characteristic findings of a recent labyrinth suppuration. In contrast with this case is that of Case 113 with nearly normal findings for after-nystagmus in spite of the history of vertigo. The case was one of right-sided chronic middle ear suppuration. Multiple turning tests were made. The figures after fifteen turns were greater than those after ten turns; however, they diminished slightly after twenty turns. Case 100, with chronic middle ear suppuration of the right side and with the history of vertigo, showed spontaneous nystagmus to the right side, more horizontal than rotatory. Also the horizontal nystagmus to the right exceeded the average normal after ten turns to the left, and exceeded considerably the horizontal after-nystagmus to the left. This would suggest a circumscribed irritative process in the right inner ear, probably located in the region of the external semicircular canal. Case 99 represents a still different type of internal ear affection in the right ear secondary to a chronic middle ear suppuration of that ear. The after-nystagmus to the right is diminished slightly. Clinically it would represent a slight or moderate degree of destruction of the semicircular canal apparatus. The after-nystagmus to the right, affected side, after ten turns is eighteen seconds, while that to the unaffected side is twenty-three seconds. In this small group of four cases no two are alike, from the standpoint of the internal ear.

In the list there are four cases, 88, 89, 102 and 110, of acute mastoid abscess. In Case 89, with mastoiditis on the right side, the after-nystagmus on that side is reduced after ten turns to ten seconds, while it is twenty-four seconds to the unaffected side. The pus was held under considerable tension on this side. There is with it a reduction of function similar to that produced in the cochlear apparatus by pressure with a Gellé bag. The figures approached normal after release of the pressure by operation on the mastoid. This same argument holds true with Case 110, which showed, prior to operation, a horizontal after-nystagmus of fourteen seconds to the right (affected side), and twenty-one seconds to the left (unaffected side). About the same figures were ascertained for the rotatory nystagmus. Both the horizontal and rotatory nystagmus were studied after ten turns. Case 88 approached the normal figures for horizontal after-turning nystagmus. Case 102 was tried after multiple turnings; the figures are relatively low, which at present I am not prepared to explain; however, they follow the rule, in that the figures increased with increased turnings.

Excluding the deafmute cases already considered, there are four cases of basal fractures involving the temporal bone. Cases 50, 87, 104, 107. Case 50 was seen eleven days after the injury, with pronounced mastoiditis and complete deafness on the right side. Vertigo was pronounced as well as a spontaneous nystagmus to the unaffected (left) side. This patient was operated shortly afterward, and at the operation a complete labyrinth operation was performed. Repeated examinations were made, with the result that the patient showed a horizontal after-nystagmus to the right, after ten turns, of eight seconds; to the left of eighteen seconds. After fifteen turns the figures increased, and after twenty they increased still more, following the general rule as ascertained from the normals; so that I am forced to conclude that what is true of two labyrinths is more or less true of one. Case 104 was likewise studied very carefully after a general surgeon had unnecessarily removed the stapes from the left ear when operating for a basal fracture. The patient immediately manifested the symptoms and signs of an acute internal ear destruction, which improved gradually. However, neither the acoustic nor vestibular functions reacted normally. In most respects the case resembled a serous labyrinthitis. This patient was turned many times and on many occasions. He was used as a demonstration case before classes. Continuous improvement was noted from time to time up to a certain point. The examinations here recorded represent the case about three months after the injury. It will be noted that the duration of the nystagmus is diminished throughout, but particularly to the left (affected) side. It is noticeable, too, that the duration for both horizontal and rotatory nystagmus increases with the number of turns. The patient showed an absence of caloric reaction early, but in time it gradually became positive. Case 87 represents one of basal fracture of one year's standing; reduction of hearing on both sides, particularly on the left. Rotatory spontaneous nystagmus to the right corresponding to destruction of the left inner ear. His after-turning nystagmus to the right is reduced. This patient continues to have vertigo and nystagmus, which suggests that the left inner ear was not completely destroyed at the time of injury, but that there is a continuous or progressive destruction which is not yet complete. This is supported by the entire findings. Case 107 represents a long standing one of basal fracture, with loss of hearing and probable loss of vestibular function. Unfortunately, the caloric test was not made, the patient objecting to further tests and failing to report later.

There was one case, 115, of ocular vertigo relieved by glasses. He presented normal findings and was well balanced to the two sides after ten turns.

There were two cases of otosclerosis, 91 and 92. They both presented the usual findings of marked impairment of hearing on both sides with tinnitus. Case 91 had vertigo. Case 92 had not. There was apparent increase of reaction to the right ear from turning to the left in Case 91, and a pronounced reduction in both ears in Case 92.

There was one case of moderately acute otitis media which improved promptly after a free paracentesis. He was examined several weeks after recovery, so that he might have been, except for the history, classed among the normals. His after-turning nystagmus to the two sides was above the average for normals, but not in excess of the high figures which are rarely found in normals. After fifteen turns his nystagmus was longer than after ten turns; however, the figures diminished somewhat after twenty turns.

I will not occupy the time and space to discuss the details of the other few odd cases; they can be readily referred to in the list.

In all, one hundred and seventeen cases were examined carefully. They include pretty nearly all the varieties of ear diseases to be met with in the average practice. I hope to increase the number of experiments in the future, but I do not see how they will materially affect the general results thus far obtained. The impetus that led me to make and collect these experiences was the fact that I had early found my results with after-turning nystagmus differed from those of the accepted authorities. Had I thought in time to make complete records of all cases, I would have had a much larger amount of material to call upon than is here presented. Since starting the work, what I have lacked in material I have tried to make up for in accuracy of the work.

Let us now compare the findings of Bárány with my own, and note in what particulars they differ:

Barany's Findings in Normals.	My Findings in Normals.
Average duration of horizontal after-nystagmus to the right after ten turns to the left was forty-one seconds.	Average duration of horizontal after-nystagmus to the right after ten turns to the left was a small fraction of a second less than twenty-four seconds.
Average duration of horizontal after-nystagmus to the left after ten turns to the right was thirty-nine seconds.	Average duration of horizontal after-nystagmus to the left after ten turns to the right was twenty-four seconds.
The maximum duration for horizontal after-nystagmus to the right after ten turns to the left was ninety-eight seconds.	The maximum duration for horizontal after-nystagmus to the right after ten turns to the left was twenty-nine seconds.
The maximum duration for horizontal after-nystagmus to the left after ten turns to the right was one hundred and twenty seconds.	The maximum duration for horizontal after-nystagmus to the left after ten turns to the right was thirty-one seconds.
Bárány makes mention of seeing one case with no nystagmus after ten turns, and uses this as his minimum for after-turning nystagmus.	I have observed two cases with apparently no nystagmus, but upon repeating the test and turning the patient at a more rapid speed and examining carefully, I was able to note and time the nystagmus, when it was found to be of relatively short excursions and lasted the usual length of time.

In sixty individuals examined on different days, he observed horizontal after-nystagmus averaging forty seconds. Maximum, eighty seconds; minimum, twenty-five seconds. He found only one-fifth of the cases showing consistently the same findings, which is the negative way of putting it that four-fifths showed variable findings.	I have examined a fewer number of cases after such methods, and have found the average, after ten turns for horizontal after-nystagmus, to equal about twenty-four seconds. I have never found anything like the variations that Bárány has. The greatest variations were but a few seconds, and these could readily have been explained on the ground of faulty observations.
In a number of cases where he had tried both rapid and slow turnings, he has found that after ten rapid turns the nystagmus lasted from eighteen to nineteen seconds. While after slow turns, from twenty-six to thirty seconds. In one-half the cases he found the rapid turns produced a longer after-nystagmus than slower turns.	I have tried patients with both rapid and slow turnings, with the result that the duration is apparently less after slow than after rapid turns, and consistently so. However, most careful tests will show them to be about equal in duration. The difference is more apparent than real, for the reason that after slow turning the nystagmus is less intensive than after rapid turning, hence less noticeable and more difficult to determine the moment of cessation.
He claims that with a great number of persons the horizontal nystagmus after twenty, thirty, forty, fifty and sixty turns was the same as after ten turns; adding that it appears that in the average ten turns show the höchsten Werte (highest worth, value or rate), while with a greater or less number of turns the amount diminishes.	I have not examined many persons after as many as thirty, forty, fifty and sixty turns, for the reason that they are hard to get. So I had to be the subject myself many times. I am, therefore, not in the position to match Bárány in material. I have not found the after-turning nystagmus after ten turns to be of the höchsten Werte in any sense in which the expression may be interpreted. For instance, I have found the nystagmus to increase consistently in duration between ten and twenty turns. Furthermore, I find less variations after twenty than after ten turns, showing, therefore, that the results are more reliable after twenty turns than after ten turns.
He claims that the amount of after-turning nystagmus after twenty turns is about equal to that after five turns.	I have never in my experience found this to be true in a single case.

He claims that after twenty or more turns there appears, not infrequently, a nach-nach-nystagmus (after-after-nystagmus) in the direction opposite to the after-nystagmus, lasting sometimes a minute.

Concerning dancers, he claims that with those who habitually turn to the right the duration of the horizontal turning nystagmus to the left falls considerably shorter than that to the right.

I have never seen an after-after-nystagmus in many hundred examinations, including those in my list. I do not believe it exists, and will not believe it until it is shown to me.

I was fortunate enough to obtain a whirling dervisher, with the stage name of Hash Hash, who was a left turner. I examined him on many occasions, and found that his duration of nystagmus was consistently the same on several occasions. The figures were reduced for both horizontal and rotatory nystagmus to both sides, and more so to the right. This one case supports the claims of Bárány. (Case 10 in the list.)

Concerning the theory that Bárány has to offer in explanation of his variable findings, I have only to say that I cannot accept it. I do not see how his findings can possibly be right without my findings, which have been carefully undertaken, supporting his; and if his findings are wrong, they being his premise, then must his conclusions be false. It does not seem likely that nature should provide us with definite centers to carry on such pranks in physiologic behavior as are accredited by Bárány, and that my findings, which are fairly consistent, should be false.

Bárány in his efforts to find a relationship between the semicircular canals and the eye muscles, neglects a certain precaution that he himself mentions when he advises the use of opaque glasses in studying horizontal after-turning nystagmus.

Bárány in his efforts to find "The Relationship Between the Semicircular Canals and the Eye Muscles" makes observations of horizontal after-turning nystagmus in the various positions of the visual axis—i. e., by having the patient direct his gaze in various positions. He also makes observations of nystagmus "by letting the patient look at the forefinger held at a distance of three-quarters of a meter"; all of which is in direct contradiction to the precautions laid down in his original monograph, where he advises the use of the opaque spectacles of Abels.

Together with many other Americans, it was my good fortune to have taken Bárány's course on the labyrinth, prior to and shortly after the publication of his work. At that time the opaque spectacles were not used on all occasions in timing after-turning nystagmus. Many tests were made exactly after the method mentioned by him in the proceedings of the Ninth International Otological Congress—that is, by having the patient look at his forefinger held three-quarters of a meter from the patient, both in front of and to the sides of the patient. He will never be able to determine the relationship between the eye muscles and semicircular canals after this manner, for the reason that as soon as he permits the patient to direct his eyes away from the middle line to one or the other side, no longer can there be a purely semicircular canal reflex, but this reflex plus tension on one or more of the extraocular muscles. A pure semicircular canal reflex nystagmus is manifested only when the patient's eye muscles are in a state of complete relaxation, if such a thing is possible. This condition is most nearly attained when the patient looks straight ahead at some distant object, and not at the finger held three-quarters of a meter from the patient.

What makes horizontal nystagmus to the left more intensive when looking to the left and less intensive when looking to the right? Surely, it cannot be that there is an actual increase or diminution of the intensity of the stimulus originating in the semicircular canals. We must look for a simpler explanation. The answer is that when looking to the left there is the stimulus in the canals which calls forth the reflex plus something else, and that when the patient looks to the right there is the stimulus in the canals which calls for the reflex minus something else. This plus and minus element is found in that normal physiologic nystagmus, or the tendency to it, to the left when looking to the left, and to the right when looking to the right; upward when looking upward, etc. It is well known that the average individual will show a horizontal nystagmus to the left when looking intently to the left, etc. In some cases, instead of a horizontal nystagmus, it may become rotatory in the extreme lateral positions of the eyes. The tendency toward rotatory nystagmus becomes greater the more the eyes are directed away from the horizontal position. I may go a step farther to prove that this physiologic nystagmus, or the tendency to it, plays an important rôle in influencing the character and intensity of the socalled vestibular nystagmus. For instance, I have repeatedly observed that a relatively intensive horizontal after-nystagmus to the left may be completely annulled by having the patient look strongly to the right. By having the patient look still stronger to the right, the physiologic element overbalances the vestibular nystagmus to the left, when he exhibits a purely rotatory nystagmus to the right. If you have not consumed too much time in your experiment thus far, you may be able to elicit a return of horizontal nystagmus to the right by having the patient look straight ahead. The character and direction of the vestibular nystagmus may be further influenced in the side positions in cases showing muscle imbalance, particularly when that imbalance is due to paretic conditions of one or more of the eye muscles. It is well known among oculists that in cases with eye muscle paresis the patient will manifest a rhythmic nystagmus more pronounced when looking in the direction corresponding to the action of the weak eye muscle than when looking in the opposite direction.

I cannot help but attribute Bárány's irregular findings, as compared to my own, as due to his technic, which I believe is faulty, rather than to actual irregularities in the behavior of the reflexes.

In his "Table for Differential Diagnosis of Mentioned Diseases," page 276, Proceedings of Ninth International Otological Congress, he indicates with arrows the direction of spontaneous nystagmus in the column, suppuration of the right labyrinth since twenty-one days and elsewhere, that the patient has spontaneous nystagmus to the right as well as to the left. A nystagmus to the right in such a condition is impossible unless the patient looks to the right. This nystagmus to the right, when looking to the right, is not in the least way connected with the patient's labyrinthine condition. It is the same physiologic nystagmus that a normal person may manifest. If we have the patient look straight ahead the nystagmus to the right would not be present. That Bárány has included it among his other findings in labyrinth suppuration only supports my previous contention, that he lays more stress upon the side positions than the straight forward position of the visual axis. He might answer by saying that he compares the intensity of the nystagmus to the two sides, and to aid him in accuracy at obtaining the same angle deviation, he uses his fixation apparatus. My reply to this would be that to ascertain the exact angle to the two sides takes some time, and during the interim one must carry in his mind the exact intensity of the nystagmus to the side previously examined, in order to make comparison; and comparisons that depend on memory ever so short cannot be as exact as those made simultaneously. Another objection is that the instrument must be tried carefully beforehand to see that zero corresponds exactly with the straight ahead position of the eyeballs. Furthermore, the proximithy of the instrument to the eyes causes a considerable degree of convergence (action of the interni muscles), which must of necessity interfere with the free action of the vestibular reflex. Finally, before the findings in the lateral positions of the eyes can be accepted in any particular case, a careful examination of the eye muscles must be made to eliminate possible heterophorias or paresis.

In his table there is no mention made of his having noted the spontaneous nystagmus that occurs when looking straight ahead; while with me no other nystagmus is of value from the standpoint of the semicircular canals. A nystagmus which is elicited by turning movements of the eyes belongs to the eye muscles. If the intensity of the nystagmus balances in opposite directions, I accept it as physiologic; when they do not, I accept it as pathologic, and the problem then resolves itself down to the isolation of the particular muscle or muscles involved.

I might add more arguments to my case, but they would be only corroborative of what has already been said. Accordingly, I will rest my case.

Case 1.—W. L., male, 28 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 11½ seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to right 25 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 26½ seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 2.—W. H. B., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 3.—C. P. W., male, 42 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 4.—A. H. C. R., male, 45 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 5.—H. A. L., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 25½ seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 6.—J. L., male, 13 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 7.—G. W. M., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 17 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 8.—A. V. M., female, 45 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 14½ seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 9.—A. K. M., male. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after turning five times to the right, with head erect, horizontal nystagmus to the left 15 seconds. After turning ten times to the left, with head erect, horizontal nystagmus to the right 21 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 22 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 10.—H. H., male, 45 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, no nystagmus to the right; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 12 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 11 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 13 seconds. After fifteen turns to the left, with head inclined forward about ninety degrees, rotatory nystagmus to the right 12 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. After twenty turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after twenty turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 4½ m.a., rotatory nystagmus to the left. Left ear, cathode 4½ m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Dervisher dancer, left turner, generally turns 118 times in a minute, has turned 150 times in a minute; fourteen years ago turned five hours and ten minutes without stopping. The nystagmus is more apparent to the left, making longer and quicker movements.

Case 11.—G. C. W., male, 26 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After turning ten times to the left, with head erect, horizontal nystagmus to the right 17 seconds; after turning ten times to the right, with head erect, horizontal nystagmus to the left 18 seconds. After turning fifteen times to the left, with head erect, horizontal nystagmus to the right 17 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 12.—G. H., male, 24 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. Aften ten turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 13.—E. La B., female, 21 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 14.—W. W. R., male, 32 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 15.—M. B., female, 16 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmas: After five turns to the left, with head erect, horizontal nystagmus to the right 6 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 6 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 9 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 18 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. After twenty turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 21 seconds; after twenty turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 7½ m.a., rotatory nystagmus to the right; anode 4½ m.a., rotatory nystagmus to the left. Left ear, cathode 4¾ m.a., rotatory nystagmus to the left; anode 7½ m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Normal case.

Case 16.—Miss B., female, 20 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after five turns to the right, with head erect, horizontal nystgamus to the left 15 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. On another occasion: After five turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the righ 28 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 2 m.a., rotatory nystagmus to the left. Left ear, cathode 2 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Normal case.

Case 17.—L. P., male, 24 years of age. No vertigo, tinnitus in both ears, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings: Right ear, membrane opaque, slightly retracted, chalk deposit posterior half; left ear, same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic test not taken. Caloric test not taken. Remarks: Came because of tonsillar condition and repeated attacks of acute secretory catarrh, and examination made during one of these attacks.

Case 18.—F. C., male, 23 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 4 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 4 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Right-sided Bell's palsy.

Case 19.—M. G., female, 18 years of age. Vertigo present off and on for several years, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction: Right ear, cathode 7 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 6½ m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Eye muscles half a degree of exophoria and half a degree of right hypophoria.

Case 20.—F. L., male, 40 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 13 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 35 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 21.—T. N. H., male, 20 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds.

After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic test not taken. Caloric test not taken. Remarks: Normal case.

Case 22.—L. P. B., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 29 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 32 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 36 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 36 seconds. Galvanic test not taken. Caloric test not taken. Remarks: Normal case.

Case 23.—D. D. C., female, 26 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 29 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 24.—J. McC., male, 23 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 25.—M. P., female, 16 years of age. Vertigo at times, tinnitus, buzzing sound in left ear, no spontaneous nystagmus. Hearing tests, impairment both ears, more in the left. Weber to the left. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, membrane slightly retracted and opaque. Left ear, moderate profuse brown offensive discharge. Large perforation anterior inferior quadrant. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: One-sided middle ear suppuration.

Case 26.—W. B., male, 23 years of age. Vertigo had been present, but not now. No tinnitus, no spontaneous nystagmus. Hearing moderately reduced. Worse in right ear. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, perforation with discharge. Left ear, dry perforation. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Bilateral chronic middle ear suppuration.

Case 27.—W. S. B., male, 35 years of age. Vertigo present, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case proved to be one of ocular vertigo, which was corrected by lenses.

Case 28.—E. K., female, 12 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, fair both sides. Weber indifferent. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. C_1 not heard on the right and shortened on the left. Otoscopic findings: Right ear, discharge. Left ear, discharge very pronounced, canal narrowed. After-turning nystagmus: After five turns to the left, with head erect, no nystagmus to the right; after five turns to the right, with head erect, no nystagmus to the left. After ten turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after ten turns to the right with head erect, horizontal nystagmus to the left 14 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. At another occasion: After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Bilateral chronic middle ear suppuration.

Case 29.—C. W., male, 25 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, hearing reduced both sides. Weber to the right. Schwabach lengthened on both sides. Rinne negative on both sides. C_1 shortened on both sides, C_4 normal on both sides. Otoscopic findings: Right ear, membrane intact, secretion showing behind the lower half of the membrane with a niveau line indicating the level of the secretion. Left ear, membrane red, bulging slightly, perforation in the anterior inferior quadrant. Secretion somewhat thin, moderate amount. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 29 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. Galvanic test not taken. Caloric test not taken. Remarks: More physiologic nystagmus to the right when looking to the right than to the left when looking to the left. Left acute middle ear suppuration, right secretory catarrh.

Case 30.—R. W., female, 8 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing fair on both sides. Weber to the right. Schwabach very slightly lengthened on the right and on the left. Rinne neutral, neither positive nor negative on both sides. Otoscopic findings: Right ear, normal. Left ear, attic perforation. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken.

Case 31.—R. W., male, 9 years of age. Vertigo, light attacks on rare occasions, no tinnitus, no spontaneous nystagmus. Hearing tests, very marked reduction both sides. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. C_1 shortened on the right and on the left. C_4 shortened on the right and on the left. Otoscopic findings: Right ear, slight secretion, hammer handle visible, also long process of the incus. Polyps on inner wall. Left ear, profuse, thick, yellow, offensive discharge. Remnants of membrane in front, hammer handle remains. Granulations in tympanic cavity. Deep opening in niche of the oval window. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction, cold water both sides positive. Remarks: Bilateral chronic middle ear suppuration left side, tuberculous mastoiditis.

Case 32.—C. B. C., male, 27 years of age. No vertigo, occasional ringing noises, no spontaneous nystagmus. Hearing tests, moderate impairment, worse on left side. Weber to the left. Schwabach shortened on the right and on the left. Rinne positive on both sides. Otoscopic findings: Right ear, membrane intact, hammer handle visible. Left ear, total destruction of membrane. Newformed adherent to the inner wall. Head of stapes seen and stapedius muscle. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. Galvanic reaction not taken. Caloric reaction not taken.

Case 33.—L. S., male, 35 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, moderate reduction on the right, normal on the left. Weber to the left. Schwabach shortened on the right. Rinne negative on the right and on the left. C_4 shortened on the right. Otoscopic findings: Right ear, membrane slightly opaque, otherwise normal. Left ear, the same. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 23 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Right-sided seventh nerve palsy; involvement of the eighth nerve, especially the cochlear branch. Developed six months before examination.

Case 34.—S. W., male, 22 years of age. Vertigo one attack; no tinnitus, no spontaneous nystagmus. Hearing tests, reduction in both ears, pronounced on the right, slight on the left. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, slight amount of creamy discharge. Granulations in attic antrum region. Left ear, membrane opaque, normal position, small scar in the anterior superior quadrant. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction positive. Remarks: Seen in consultation with Dr. Dowling because of continued discharge after radical operation on right side.

Case 35.—W. R., female, 51 years of age. Vertigo, dizziness worse when lying down; tinnitus like roaring of the sea, no spontaneous nystagmus. Hearing tests, marked reduction on the left, normal on the right. Weber to the right. Schwabach slightly shortened on the left. Rinne positive on the left. Otoscopic findings: Right ear, membrane slightly opaque, otherwise normal. Left ear, the same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 23 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Meningitis five months previous.

Case 36.—J. E. O., female, 35 years of age. Vertigo, dizziness slight and of short duration; no tinnitus, no spontaneous nystagmus. Hearing tests: Left ear, reduced. Right ear, slightly reduced. Weber to the left. Schwabach shortened on the right and on the left. Rinne positive on the right, and neutral, neither positive nor negative, on the left. Otoscopic findings: Right ear, membrane normal color but slightly retracted. Left ear, bulging and red membrane, no perforation. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 22 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Subsequent operation for mastoiditis left side.

Case 37.—F. G., male, 24 years of age. Has vertigo, no tinnitus; spontaneous nystagmus rotatory to the right looking straight ahead. Hearing tests, complete deafness on the left. Weber to the right. Schwabach normal on the right and shortened on the left. Rinne positive on the right and negative on the left. C_1 not heard on the left and C_4 not heard on the left. Otoscopic findings: Right ear, membrane slightly cloudy, otherwise normal. Left ear, membrane totally destroyed. Polyps from attic antrum region. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 7 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 7 m.a., no reaction. Left ear, cathode 7 m.a., no reaction; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the left ear negative. Remarks: One-sided labyrinth destruction. Chronic middle ear suppuration left side, cholesteatoma, fistula of the external semicircular canal, meningitis. Radical and labyrinth operation. Cured.

Case 38.—L. H., male, 33 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in the right ear by all tests. Otoscopic findings: Right ear, destruction of right membrane with offensive discharge. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. Galvanic reaction: Right ear, cathode 7 m.a., rotatory nystagmus to the right; anode 3 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction, cold water in right ear negative. Remarks: Chronic middle ear suppuration right side, with cholesteatoma, fistula of the external semicircular canal and labyrinth suppuration.

Case 39.—F. K., male, 34 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in the left ear by all tests. Otoscopic findings, left membrane destroyed, offensive discharge. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 7 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 8 m.a., no reaction. Left ear, cathode 8 m.a., no reaction; anode 8 m.a., no reaction. Caloric reaction, cold water to the left ear negative. Remarks: Chronic middle ear suppuration on the left side with labyrinth sequestration. Left-sided facial paralysis.

Case 40.—R. W., male, 27 years of age. Has vertigo, no tinnitus, spontaneous nystagmus, rotatory to the right when looking straight ahead. Hearing tests, deaf on left side by all tests. Otoscopic findings: Destruction of the left membrane with discharge. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 35 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 19 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 7 m.a., no reaction. Left ear, cathode 7 m.a., no reaction; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Chronic middle ear suppuration left side with cholesteatoma and destruction of the labyrinth.

Case 41.—G. G., male, 37 years of age. Has vertigo with vomiting, no tinnitus, no spontaneous nystagmus. Hearing tests, deafness on the right side. Otoscopic findings, right membrane destroyed with secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 5 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 10 seconds. Galvanic reaction: Right ear, cathode 12 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 6 m.a., rotatory nystagmus to the left; anode 12 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the right ear negative. Remarks: Chronic middle ear suppuration right side with destruction of the labyrinth and facial paralysis.

Case 42.—W. P., male, 34 years of age. Has vertigo with vomiting, no tinnitus, spontaneous nystagmus, slight rotatory nystagmus to the left when looking straight ahead. Hearing tests, deaf in the right ear by all tests. Otoscopic findings, destruction of the right membrane. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 6 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 23 seconds. Galvanic reaction: Right ear, cathode 16 m.a., no reaction; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 14 m.a., no nystagmus. Caloric reaction, cold water to the right ear negative. Remarks: Right chronic middle ear suppuration with labyrinth destruction. Radical operation performed together with labyrinthine drainage.

Case 43.—A. K., female, 40 years of age. Has intense vertigo, no tinnitus, spontaneous nystagmus, rotatory nystagmus to the right when looking straight ahead. Hearing tests, complete deafness in left ear. Otoscopic findings, left membrane totally destroyed, offensive discharge. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. Galvanic reaction: Right ear, cathode 2 m.a., rotatory nystagmus to the right; anode 12 m.a., rotatory nystagmus to the left. Left ear, cathode 12 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the left ear negative. Remarks: Chronic middle ear suppuration left side with labyrinth suppuration. Meningitis. Left facial palsy. Radical and labyrinth operations.

Case 44.—E. K., female, 46 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in the right ear by all tests. Otoscopic findings, total destruction of the right membrane with offensive discharge. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 7 seconds; after ten turns to the right, with head inclined forward about 90 degrees; rotatory nystagmus to the left 14 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 3 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the right ear negative. Remarks: Chronic middle ear suppuration, right side, with cholesteatoma and destruction of the labyrinth. Carcinoma.

Case 45.—W. L. B., male, 31 years of age. Vertigo marked. One spontaneous attack in office associated with rotatory nystagmus to the left, tinnitus in right ear, spontaneous nystagmus present off and on, variable in intensity, always to the left. Hearing tests, right markedly diminished. Weber to the left. Schwabach shortened on the right, normal on the left. Rinne negative on the right and positive on the left. Otoscopic findings: Right ear, membrane slightly cloudy and retracted. Left ear, same, but less pronounced. After-turning nystagmus: After ten turns to the left, with head erect; horizontal nystagmus to the right 13 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Syphilitic lesion both sides, more pronounced right.

Case 46.—S. A., female, 60 years of age. Has vertigo, has tinnitus, worse in left ear, no spontaneous nystagmus. Hearing tests, fair right ear, deaf in left ear. Otoscopic findings: Right ear, slightly opaque. Left ear, membrane slightly cloudy and retracted. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Labyrinth suppuration secondary to acute middle ear suppuration. Seen in consultation with aurist, who treated the case conservatively. Examination was made three months after onset of the trouble. A few weeks after this patient was able to be around.

Case 47.—D. L., male, 38 years of age. Formerly severe attacks of vertigo, but at present only occasional slight attacks; tinnitus, buzzing sound, worse at night, no spontaneous nystagmus. Hearing tests, reduction in both ears, more in the left. Weber to the right. Schwabach shortened on the right and on the left. Rinne positive on the right and on the left. Otoscopic findings: Right ear, membrane slightly cloudy, normal mobility. Left ear, same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 22 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right: anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Sequela of eighth nerve neuritis of questionable origin, possibly syphilis. Wassermann negative; the patient gave history of infection (chancroid with bubo).

Case 48.—R. McC., female, 39 years of age. Vertigo pronounced with vomiting, has tinnitus, spontaneous nystagmus rotatory to the right when looking straight ahead. Hearing tests, complete deafness left side by all tests. Otoscopic findings: Right ear, normal. Left ear, membrane intact, dull and opaque. Anatomic details impossible. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 9 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 4 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 5 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 1 second; after five turns to the right, with head inclined forward about 90 degrees, no rotatory nystagmus to the left. After ten turns to the left, with head inclined forward about 90 degres, rotatory nystagmus to the right 8 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 6 seconds. After fifteen turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 6 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 10 m.a., cessation of nystagmus. Left ear, cathode 10 m.a., no reaction on the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction negative. Remarks: Rapid labyrinth destruction of the left side following a few days after the onset of acute middle ear suppuration.

Case 49.—G. L., male, 52 years of age. Vertigo pronounced at onset, but none at present; tinnitus came on suddenly eight weeks ago, slight at present time; spontaneous nystagmus rotatory to the left. Hearing tests, complete deafness in right side, left side normal. Otoscopic findings negative. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 7 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction present both sides, but diminished to the right. Remarks: Gave history of syphilitic infection many years ago. At time of test, blood pressure 200.

Case 50.—W. A. S., female, 47 years of age. Has vertigo, tinnitus pronounced, no spontaneous nystagmus. Hearing tests, marked reduction in both ears. Weber indifferent. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: A slight opacity of membrane, but no retraction both ears. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Late lues. Evidences of old interstitial keratitis.

Case 51.—G. D., male, 25 years of age. Vertigo pronounced, no tinnitus; spontaneous nystagmus, rotatory to the left looking straight ahead. Hearing tests, complete deafness on the right side. Otoscopic findings: Right ear, external canal swollen, pulsating discharge. Anatomic details not seen. Swelling over mastoid. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 8 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 9 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. Galvanic reaction not taken. Caloric reaction negative on the right side. Remarks: Fractured skull involving base. Seventh nerve paralysis complete. At time of operation fractured fragment of temporal bone involving inner ear was removed.

Case 52.—F. E. H., female, 6 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in both ears by all tests. Otoscopic findings: Right ear, membrane opaque and retracted; no perforation, no scars. Left ear, same. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. After twenty turns to the left, with head erect, and after twenty turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Ophthalmoscopic examination showed excessive connective tissue on disc, which, together with the history and findings, suggested meningitis at the age of three years.

Case 53.—J. M. K., male, 54 years of age. Vertigo pronounced off and on, tinnitus present, no spontaneous nystagmus. Hearing tests, deafness almost complete in both ears. Otoscopic findings: Right ear, membrane destroyed, no discharge. Left ear, membrane destroyed, large polyp from antrum region, scanty secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 8 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. Galvanic reaction not taken. Caloric reaction, ice water for three minutes in left ear, mild rotatory nystagmus to the right. Remarks: Unsteady gait, moderate destruction of both acoustic and vestibular apparatus in both ears.

Case 54.—J. O., male, 13½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, both sides normal. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf following meningitis at the age of one year.

Case 55.—A. J., male, 15½ years of age. No vertigo, tinnitus in the left, spontaneous nystagmus, slight rotatory nystagmus to the right when looking straight ahead. Hearing tests, complete deafness both sides. Otoscopic findings, both sides membrane normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 6 seconds; after ten turns to the right, with head inclined forward about 90 degrees, no nystagmus to the left. Galvanic reaction: Right ear, cathode 1 m.a., rotatory nystagmus to the right; anode 16 m.a., no reaction on the left. Left ear, cathode 12 m.a., no reaction on the left; anode 12 m.a., no reaction on the right. Caloric reaction not taken. Remarks: Deaf since five years of age, following meningitis.

Case 56.—M. B., female, 14½ years of age. No vertigo, tinnitus left side, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings: Membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 14 m.a., no reaction on the right; anode 14 m.a., no reaction on the left. Left ear, cathode 14 m.a., no reaction on the left; anode 14 m.a., no reaction on the right. Caloric reaction not taken. Remarks: Deaf since three years of age, following meningitis.

Case 57.—L. K., female, 9 years of age. No vertigo, tinnitus both sides, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings: Right ear, membrane slightly opaque. Left ear, normal. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since eight years of age, following meningitis.

Case 58.—S. P., female, 12½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membrane both sides opaque. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since two years of age, following meningitis.

Case 59.—M. W., female, 9½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, slight remains of hearing on both sides. Otoscopic findings, both sides normal membranes. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf following meningitis.

Case 60.—M. G., male, 22 years of age. No vertigo, tinnitus both sides, more to the left, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membrane normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 10 m.a., rotatory nystagmus to the right; anode 12 m.a., rotatory nystagmus to the left. Left ear, cathode 12 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since twelve years of age, following meningitis.

Case 61.—G. S., male, 10 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, normal membranes both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since five years of age, following a long fever. (Meningitis?)

Case 62.—N. K., male, 17 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Ostoscopic findings, both sides normal. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 18 m.a., no reaction to the right; anode 18 m.a., rotatory nystagmus to the left. Left ear, cathode 18 m.a., rotatory nystagmus to the left; anode 18 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 63.—A. N., male, 13½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing very greatly reduced both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction: Right ear, cathode 7 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 9 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 64.—A. R., male, 15 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 25 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 22 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Deaf since birth.

Case 65.—R. E., male, 14 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes opaque and retracted both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 14 m.a., no reaction to the right; anode 14 m.a., no reaction to the left. Left ear, cathode 14 m.a., no reaction to the left; anode 14 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 66.—A. N., male, 13½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, both sides normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 10 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Deaf since birth.

Case 67.—O. F., female, 12½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 17 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 68.—M. W., female, 15½ years of age. No vertigo, tinnitus both sides, no nystagmus. Hearing tests, very slight remains on both sides. Otoscopic findings, right membrane slightly opaque, left normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 10 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 6 m.a., rotatory nystagmus to the left; anode 10 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth. Sister to O. W. (Case 69), also deaf since birth.

Case 69.—O. W., female, 14 years of age. No vertigo, tinnitus to the right, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 6 m. a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth. Sister to M. W. (Case 68), also deaf since birth.

Case 70.—A. S., male, 14½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membrane opaque both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 19 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 71.—A. E., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, slight remains of hearing. Otoscopic findings, membrane normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction;

anode 12 m.a., no reaction. Left ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since birth.

Case 72.—V. H., male, 16½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, total destruction both membranes with granulations in tympanic cavity. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 8 m.a., rotatory nystagmus to the right; anode 11 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 8 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since two years of age, following measles.

Case 73.—A. W., female, 13½ years of age. No vertigo, no tinnitus to the left, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, total destruction of the membranes, both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 17 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 14 seconds. Galvanic reaction: Right ear, cathode 10 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 10 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since six years of age, following measles.

Case 74.—F. K., male, 13 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing greatly reduced on both sides. Otoscopic findings, membranes retracted and opaque both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 4 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 5 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 3 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Very deaf since three years of age from unknown cause.

Case 75.—M. B., male, 13½ years of age. No vertigo, no tinnitus, spontaneous nystagmus, slight rotatory nystagmus to the left when looking straight ahead. Hearing tests, very slight remains both sides. Otoscopic findings, membranes thickened and opaque both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction, unable to examine because of nervous condition. Caloric reaction not taken. Remarks: Deaf since two years of age from a blow on the head.

Case 76.—K. K., male, 11½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, very greatly reduced in the right ear, complete deafness in the left. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since five years of age, from a blow on the head.

Case 77.—J. B., male, 13½ years of age. No vertigo no tinnitus, no spontaneous nystagmus. Hearing very greatly reduced on both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 10 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 12 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since three years of age from a blow on the head.

Case 78.—L. A., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, very slight remains both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 8 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 9 seconds. Galvanic reaction: Right ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Left ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since one year of age from a blow on the head.

Case 79.—F. L., male, 10 years of age. No vertigo, tinnitus both sides, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes slightly opaque and retracted both sides. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus, either to the right or to the left. Galvanic reaction: Right ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Left ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since eight years of age, following a fall on the back of the head.

Case 80.—M. L., male, 14 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 14 m.a., no reaction; anode 14 m.a., no reaction. Left ear, cathode 14 m.a., no reaction; anode 14 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since three years of age, following scarlet fever.

Case 81.—E. U., male, 16 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Left ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since three years of age, following typhoid fever.

Case 82.—H. N., female, 15 years of age. No vertigo, tinnitus to the left, no spontaneous nystagmus. Hearing tests, very slight remains of hearing on both sides. Otoscopic findings, membranes normal on both sides. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Left ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since four years of age; cause unknown.

Case 83.—W. W., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness right side, slight remains on the left. Otoscopic findings, membranes opaque both sides, with chalk deposit on the right. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 10 m.a., no reaction; anode 8 m.a., no reaction. Left ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since one year of age, following whooping cough.

Case 84.—J. E. T., male, 64 years of age. Vertigo present, probably of ocular origin, improved with correcting lens, has tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction in both ears, more in the right. Otoscopic findings, both membranes slightly opaque and retracted. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 31 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 29 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 6 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Claims to have had cerebral hemorrhage eight years ago with vertigo, vomiting and complete loss of hearing.

Case 85.—D. M., male, 23 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, impairment of both sides, worse on the right. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings, almost complete destruction of membrane on both sides with cholesteatoma and discharge. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 33 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 32 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 22 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 26 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Chronic middle ear suppuration both sides, fistula symptom positive left side.

Case 86.—J. T. D., female, 17 years of age. Vertigo pronounced, no tinnitus, spontaneous nystagmus horizontal to the right. Hearing tests, moderate reduction on the left. Weber to the left. Rinne negative to the left, right ear normal. Otoscopic findings, left ear, moderate destruction of membrane with discharge and granulations. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 5 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction: Cold water in left ear, head erect, rotatory nystagmus to the right. Remarks: Case of fistula of the external semicircular canal. Operated for radical mastoid, healing with retention of function in the vertical semicircular canal.

Case 87.—W. S., male, 56 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, slight reduction both sides. Otoscopic findings, fairly normal membrane both sides. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 18 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 8 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Mild lesion of the eighth nerve, left side, of questionable origin.

Case 88.—J. A., male, 38 years of age. Vertigo present some months ago but not now, no tinnitus, spontaneous nystagmus to the right when looking straight ahead. Hearing tests, great reduction in both ears, more on the left. Weber to the left. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right membrane opaque and retracted. Left ear the same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Injury to head one year ago.

Case 89.—W. H., male, 20 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction on the right, left normal. Otoscopic findings, right canal narrowed, perforation of the membrane; impossible to get other anatomic details. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Acute mastoiditis. Operation.

Case 90.—J. B. D., male, 14 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction on the right side, left side normal. Otoscopic findings, extensive swelling over the right mastoid, canal narrowed, bulging of right membrane, pulsating secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after ten turns to the right, with head inclined forward about 90 degress, rotatory nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Acute mastoiditis right side.

Case 91.—D. M. B., male, 24 years of age. Vertigo present only by quick movements of the head, no tinnitus, no spontaneous nystagmus. Hearing tests, total deafness in left ear, right ear normal. Otoscopic findings, normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case of congenital deafness in left ear.

Case 92.—S. G., female, 52 years of age. Has vertigo, for the last three or four years much worse, has tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction in both ears, more on the right. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Otosclerosis with internal ear involvement.

Case 93.—A. C., female, 43 years of age. No vertigo, tinnitus pronounced, no spontaneous nystagmus. Hearing tests, very marked reduction in both sides. Weber to the left. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings, membranes normal appearance both sides, promontory pinker than normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 11 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Otosclerosis with involvement of inner ear.

Case 94.—A. J., male, 41 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, reduction of hearing in both ears, more on the right. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right canal narrowed, profuse offensive discharge, complete destruction of the membrane, attic granulations. Left ear, complete destruction of membrane, no secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 19 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction not taken. Caloric reaction positive in the right ear. Remarks: Chronic middle ear suppuration with cholesteatoma right side. Chronic adhesive process following suppuration left side.

Case 95,—M. K., female, 13 years of age. Vertigo present, no tinnitus, spontaneous nystagmus rotatory to the right looking straight ahead. Hearing tests, marked impairment of hearing on right. Schwabach shortened on the right. Rinne negative on the right. Otoscopic findings, right ear, small perforation, profuse fetid pus. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 25 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction: Right ear, cathode 1 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 1 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to right ear, rotatory nystagmus to the left. Remarks: Chronic middle ear suppuration on the right side, chronic adhesive process on the left.

Case 96.—E. C. T., female, 12 years of age. Has vertigo, tinnitus occasionally, no spontaneous nystagmus. Hearing tests, marked reduction both ears, more to the right. Weber to the left. Schwabach slightly lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, large perforation, fetid purulent secretion, inspissated mucus on inner wall. Left ear, large perforation in superior posterior quadrant. Polyps in the antrum region. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Cholesteatoma in both ears.

Case 97.—K. F., female, 22 years of age. Has vertigo occasionally, no tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction both ears, especially in right. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, complete destruction of membrane, profuse offensive discharge and cholesteatoma. Left ear, complete destruction of membrane, granulations on promontory, remnants of hammer handle adherent to the inner wall. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 23 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 22 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 3 m.a., rotatory nystagmus to the left. Left ear, cathode 3 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction, apparently negative on left side, positive on the right. Remarks: Bilateral chronic middle ear suppuration.

Case 98.—F. S., male, 27 years of age. Vertigo has been present but description of it is poor, tinnitus both sides, no spontaneous nystagmus. Hearing tests, marked reduction. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, small part of membrane remaining, scant, dark, offensive discharge, ossicles gone, granulations present. Left ear, slight, offensive discharge, canal full of polyps. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 9 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 6 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 8 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 8 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. After fifteen turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. After twenty turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after twenty turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 5½ m.a., rotatory nystagmus to the left. Left ear, cathode 4½ m.a., rotatory nystagmus to the left; anode 4½ m.a., rotatory nystagmus to the right. Caloric reaction: Cold water to the right ear, rotatory nystagmus to the left; cold water for three minutes in left ear, pronounced rotatory nystagmus to the right. Remarks: Bilateral chronic middle ear suppuration.

Case 99.—A. D., male, 33 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing reduced in the right ear. Schwabach lengthened on the right. Rinne negative on the right. Left ear normal. Otoscopic findings, right ear, membrane entirely destroyed, thick yellow, offensive discharge, cholesteatoma in the attic region. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 23 seconds. Galvanic reaction not taken. Caloric reaction: Right ear, positive. Remarks: Unilateral chronic middle ear suppuration.

Case 100.—L. U., male, 13 years of age. Vertigo present, tinnitus present, no spontaneous nystagmus. Hearing tests, marked reduction of the perceptive type. Otoscopic findings, offensive discharge in right ear. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 6 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. Galvanic reaction not taken. Caloric reaction: Cold water to the right ear negative. Remarks: Unilateral chronic middle ear suppuration with destruction of the labyrinth.

Case 101.—L. H., female, 34 years of age. Vertigo present with vomiting, no tinnitus, spontaneous nystagmus, rotatory to the right looking straight ahead. Hearing tests, great impairment of the right side. Weber to the right. Schwabach slightly shortened on the right. Rinne negative on the right. Otoscopic findings, membrane totally destroyed, hammer handle retracted, inner wall thick and red, secretion scant. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 52 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction: Cold water to the right ear, rotatory nystagmus to the left. Remarks: Chronic middle ear suppuration right side, with acute congestion of the labyrinth.

Case 102.—H. E. F., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, reduction both ears, worse in the left. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, large heart-shaped perforation, Shrapnell's membrane intact. Left ear, complete loss of membrane, hammer handle present and retracted, but not adherent to the inner wall, promontory pale, niche of the oval window visible. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Chronic middle ear suppuration for nine years.

Case 103.—M. P., female, 8 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, hearing very much reduced on left side; right side normal. Otoscopic findings, left ear, pulsating discharge; difficult to get anatomic details because of the swollen canal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 5 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 7 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 13 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case of acute mastoiditis left side.

Case 104.—G. McM., male, 49 years of age. No vertigo, no tinnitus, spontaneous nystagmus horizontal to the right when looking straight ahead. Hearing tests, very marked reduction in hearing both sides. Weber to the right. Schwabach lengthened on the right and on the left. Rinne positive on the right and on the left. Stenger's test showed absolute deafness in left ear. Otoscopic findings: Right ear, membrane intact, picture of old catarrhal condition. Left ear, membrane intact. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after ten turns to the right, with hear erect, horizontal nystagmus to the left 10 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case of brain tumor about the cerebellar pontine angle, choked disc both sides.

Case 105.—W. H., male, 13 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction of hearing in the left ear. Weber to the right. Schwabach very short on the left. Rinne negative on the left. Otoscopic findings, left membrane torn, through which blood issued. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 17 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 19 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 7 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 17 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. After fifteen turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 24 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction: After prolonged use of cold water to the left ear, rotatory nystagmus to the right, but not intensive. Remarks: Patient brought to the hospital unconscious, with blood running from left ear. Suspected basal fracture corroborated at time of operation by general sugeon, who unnecessarily removed the stapes. Was then referred to me, at time of partial destruction of both acoustic and vestibular functions.

Case 106.—E. E., female, 43 years of age. Has dizziness when making rapid turns, tinnitus slight in the left ear, no spontaneous nystagmus. Hearing tests, slightest reduction in left ear to low tones. Weber to the left. Otoscopic findings: Right ear, membrane normal. Left ear, small chalk deposit anterior inferior quadrant. After-turning nystagmus: After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 23 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: From a neurologic standpoint, patient is a hypochondriac.

Case 107.—H. B. N., male, 43 years of age. Has vertigo, has tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction in both ears, more in the right. Weber indifferent. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right canal filled with polyps; impossible to get anatomic details. Left membrane totally destroyed, polyps covering promontory. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Chronic middle ear suppuration both sides.

Case 108.—W. U. S., male, 29 years of age. Vertigo very slight, no tinnitus, no spontaneous nystagmus. Hearing tests, totally deaf in the left side; right side normal. Otoscopic findings, both membranes intact. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Injury to the left side of the head nine years ago.

Case 109.—E. M. J., female, 54 years of age. No vertigo, tinnitus more to the right side, no spontaneous nystagmus. Hearing tests, hearing reduced both sides. Weber indifferent. Schwabach shortened on the right and on the left. Rinne positive on the right and on the left side. Otoscopic findings, both membranes slightly opaque. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 21 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Moderate degree of internal ear affection both sides, more pronounced on the right.

Case 110.—W. M., male, 75 years of age. Vertigo pronounced, no tinnitus, no spontaneous nystagmus. Hearing tests, reduction both ears, short of total on the left. Weber indifferent. Schwabach shortened on the right and on the left. Rinne positive on the right and negative on the left. Otoscopic findings, after removing cerumen, both membranes slightly retracted and opaque. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 12 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Patient had six degrees of exophoria.

Case 111.—M. B., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, impaired on right side. Weber to the right. Schwabach lengthened on the right. Rinne negative on the right. Otoscopic findings, right membrane swollen, sagging of the posterior wall. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Acute mastoiditis right side.

Case 112.—W. L. D., male, 56 years of age. Vertigo present but not recently, no tinnitus, no spontaneous nystagmus. Hearing tests, normal by all tests. Otoscopic findings, after the removal of cerumen, both membranes normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. After eight turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 19 seconds; too sick to continue. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Blood pressure 200, transient vertigo, due probably to high blood pressure.

Case 113.—L. S., female, 16 years of age. Vertigo occasionally, no tinnitus, no spontaneous nystagmus. Hearing slightly reduced, more in the right ear. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, newformed membrane, atrophic, depressed and adherent to inner wall, chalky deposit. Left ear, normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 8 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Nervous individual.

Case 114.—C. R., female, 35 years of age. Vertigo recently, tinnitus present, especially to the right, no spontaneous nystagmus. Hearing tests, marked impairment of hearing in both ears. Weber to the right. Schwabach normal on the right and slightly shortened on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, complete destruction of membrane, remnant of hammer handle in contact with swollen, reddened inner wall, round firm polyp in attic antrum region, granulations in the hypotympanum. Left ear, membrane opaque, retracted, short process prominent. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 28 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 28 seconds. Galvanic reaction not taken. Caloric reaction: Patient claimed to be dizzy during the test, but no nystagmus was noticed. Remarks: Divergent strabismus, patient fixes with left eye.

Case 115.—F. O., male, 22 years of age. Vertigo for ten days; was in bed with grippe and was dizzy the entire time, tinnitus in right ear four or five weeks for the last six months, very loud noises, no spontaneous nystagmus. Hearing tests, slight impairment both sides. Weber to the left. Schwabach normal on the right and lengthened on the left. Rinne positive on the right and negative on the left. Otoscopic findings, right membrane red and retracted, chalk deposits. Left membrane opaque, red hammer handle, increased mobility. After-turning nystagmus. After five turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 35 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 37 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 32 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Subacute otitis media both sides.

Case 116.—E. L. B., female, 21 years of age. Vertigo for two years, nauseated but does not vomit during attacks, tinnitus for two years in the right ear, no spontaneous nystagmus. Hearing normal on both sides. Otoscopic findings, posterior portion of the membrane slightly cloudy and overmobile, hammer handle normal both ears. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 13 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Vertigo was found to be of ocular origin.

Case 117.—S. P., female, 23 years of age. Vertigo occasionally, no tinnitus, no spontaneous nystagmus. Hearing tests, normal both sides. Otoscopic findings: Right ear, large chalk deposits posterior quadrant, and smaller one in anterior quadrant. Left ear, membrane atrophic, chorda tympani nerve seen. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. After fifteen turns to the left with head erect, horizontal nystagmus to the right 38 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 38 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 40 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 39 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Despite the fact that both membranes presented abnormal appearances, the hearing was normal both sides.

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XXVI.

VERTIGO: ITS CAUSES AND METHODS OF DIAGNOSIS.*

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The subject of vertigo is as old as the healing art itself, and medical literature abounds with essays on the subject. When vertigo was seen to accompany certain diseases, it was spoken of as vertigo belonging to that affection. Thus, when it appeared in the course of nephritis, it was kidney vertigo; if in association with an affection of the liver, it was hepatic vertigo, and so on. As a result, there were almost as many kinds of vertigo as there are diseases; but when the vertigo appeared independently and not in the course of some disease, it was termed idiopathic or essential vertigo. That vertigo was essentially a disturbance of equilibration was gleaned long ago, but so long as the equilibratory function itself was not clearly understood, vertigo remained in the land of mystery.

For many centuries the individual was regarded as possessing only five special senses; that is, sight, hearing, taste, smell, and touch. A great advance was made when the sixth sense —the socalled "muscle-joint-and-splanchnic sense"—came to be recognized. The recent studies of the internal ear, however, show us that there is still another, seventh special sense —the socalled "static sense." This static sense is a special sense as truly as any of the others, and corresponds to them in its general anatomic and physiologic requirements. Similar to them, it has an "end organ" for receiving stimuli, a conducting nerve for conveying sensations, and definite nerve tracts leading to brain centers where the stimuli are interpreted and translated into equilibration. A host of investigators have conducted experiments showing the effect on equilibration when the internal ear is either partly or completely destroyed. As a result of these, it is now universally recognized that the static portion of the internal ear is the chief organ of equilibration—in other words, it is the "end organ" of this seventh "static sense." To be sure, the ear is not the only organ concerned with the equilibratory function. The equilibrium of the body is maintained because of a harmonious cooperation of three senses—sight, muscle, and static sense. Of the three, however, the end organ of the static sense is of peculiar interest, because its only function is the maintenance of balance, whereas with the other two, equilibration is simply a collateral function.

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Summing up, then, we must have in mind the following:

1. Equilibrium of the body is maintained chiefly by the ear and its associated pathways and centers known as the vestibular apparatus, aided by the sense of sight and the muscle sense.

2. Vertigo is a conscious sensation perceived within the brain when perfect equilibration is interfered with.

This makes it evident that in considering the causes of vertigo, we must look for those disturbances which will affect the chief organ of equilibration—that is, the vestibular apparatus. From a medical standpoint, therefore, the most important aspect of the study of the static portion of the internal ear and its innervation is the subject of vertigo. The doctor is often confronted with cases of dizziness that tax his ingenuity to the utmost, and it is surely not overstating the fact to say that at best he can only guess as to its significance. The realization that the ear is the chief organ of balance throws a new light on its importance. Previous to the conception of the ear as an equilibratory organ, the general practitioner regarded the ear somewhat as follows: If the patient is deaf, a specialist should see what he can do to improve the hearing, and if there is any inflammation of the ear, it should receive local attention. But far more important, and yet scarcely recognized, is the part that the static portion of the ear plays in the well-being of the entire body. The aurist naturally knows of the recent advances in the study of the ear, but it might not be amiss to emphasize their importance to the general practitioner as well, because of:

1. The necessity of recognizing internal ear conditions which he would overlook if he were not acquainted with the subject.
2. The far-reaching significance of the ear as the sense organ of equilibration.
3. The value of the new ear tests in determining the cause of vertigo, no matter what its type or origin. It is with such cases that the physician comes in daily contact.

The general practitioner is familiar with conditions of the external ear, the middle ear, and the cochlear portion of the internal ear. In the external ear he encounters impacted cerumen, foreign bodies, eczema, and furunculosis. In the middle ear he has to deal with various types of deafness and with inflammatory conditions. Affections of the cochlea manifect themselves by impaired hearing and noises in the head. The attention of the physician, therefore, is immediately called to the ear—in fact, the patient himself recognizes that he has an ear condition. But whereas all these are of interest from a local standpoint only, the static portion of the ear, on the contrary, is part and parcel and servant of the whole organism. A big, strong, healthy man is unable to stand up or even sit up in bed because of a slight congestion of the internal ear; perhaps the pathologic process is no larger in extent than the head of a pin, and yet the man is entirely incapacitated. Just as affections of the cochlea are manifested by impaired hearing and tinnitus, the disturbance of the static portion of the internal ear produces dizziness, staggering, nausea, vomiting, and diarrhea; but whereas tinnitus and deafness at once point to the ear, we venture to say that when confronted with dizziness and nausea, the general practitioner is not apt to suspect the ear. The cases of vertigo from ear conditions with nausea, vomiting, and retching, that are treated with "stomach powders" are legion; and it cannot be too strongly emphasized that vertigo from whatever cause can result only from an affection of the vestibular apparatus directly, so that the condition of the ear and its associated pathways should be thoroughly investigated.

Vertigo may be of two kinds:

1. Experimental or induced vertigo.
2. Vertigo resulting from disease.

Experimental or induced vertigo results when the equilibratory end organ is powerfully stimulated by artificial means. Normally every conceivable motion of the body—be it in a linear or rotary plane—causes a movement of the lymph in some portion of the static labyrinth, either in the saccule or utricle, or in the semicircular canals, which in turn stimulates the sensitive hair cells of the end organs placed there. The impulses thus generated are carried along the vestibular nerve pathways to their respective centers. Inherited memory dating back for countless ages, as well as innumerable repetitions of this sort during the individual's lifetime, result in a correct interpretation as to the meaning of all these stimuli, and the person learns to know that a certain stimulus or set of stimuli always means a certain kind of motion. The individual has also learned that this sort of information is reliable and true and in accordance with fact. He is, therefore, not in the least disturbed by them. Should the lymph in the labyrinth, however, be made to move violently by artificial means, such as can be done by turning an individual in a revolving chair or douching the ear with cold or hot water, he would then be getting information which is not true to fact. For example, after he is turned in a chair and suddenly stopped, the endolymph still continues circulating, conveying the information to the brain that he is moving, whereas, as a matter of fact, he is not. Here, then, he has information which is not true to fact, and he therefore has vertigo. While he was being turned, the endolymph also moved and conveyed to him the sensation of motion, but that produced no vertigo because it was in accordance with actual facts—his previous experience assured him that he actually was moving. Cooling or heating a portion of the labyrinth by means of douching also produces motion of the lymph. Since, however, the individual is sitting quietly in the chair all this time, such information is not in accordance with fact, and therefore results in vertigo. This induced or experimental vertigo is of great service in the study of the integrity of the pathways responsible for this vertigo, and can be made of great clinical value.

Vertigo resulting from disease is entirely different. It results whenever there is an interference with the perfect balance between the special static organs on each side. Both ears always act in unison, and are constantly sending out an equal flow of impulses to the musculature of the body, insuring perfect equilibration. When disease destroys one side completely or partially, or produces an irritation—be it in the ear itself or along any pathway leading from it—there immediately results an inequality of tonic influence between the two sides, with the resulting disturbance in equilibration and vertigo. The important thing to remember is that it is always a disturbance of the ear and its associated pathways that can result in vertigo, and nothing else. When remote organs produce vertigo, it is only because they definitely influence the ear mechanism.

Vertigo may be caused by:

1. Lesions within the internal ear, such as labyrinthitis of the various types, or effusions or hemorrhage within the internal ear. Inflammatory conditions of the middle ear, for instance, acute otitis media or mild inflammation of the inner ear itself, produce only irritative effects, if any, so that the patient suffers from more or less vertigo as long as the acute state of the congestion lasts, the vertigo vanishing with the disappearance of the inflammation. On the other hand, slow degenerative changes within the labyrinth, or sudden destruction of the whole or part of one labyrinth, are accompanied by marked vertigo, nausea, and vomiting, and loss of equilibration. Sudden destruction of the whole or part of the labyrinth may be produced, rarely by trauma, but usually by hemorrhage or serous effusion into it, and it may occur in diabetes, Bright's disease, or in any condition where the vascular system becomes affected. It is this class of cases that exhibits the socalled Meniere's symptom complex. The hearing in these cases is usually markedly affected, if not altogether gone, and the condition is characterized by repeated attacks with a suddenness of onset, the violence of the symptoms quickly reaching a climax and then gradually subsiding, and all of it disappearing when the brain centers have learned to compensate.

2. Toxemias, affecting the ear or other portion of the vestibular apparatus, such as ptomain poisoning, constipation, alcoholism, poisoning by chemicals, as, for example, lead poisoning, nephritis, gout, rheumatism, syphilis, and the toxemia of infectious fevers, such as scarlatina, typhoid fever, and mumps.

3. Definite lesions along the pathways from the ear within the brain itself, such as tumor, hemorrhage, thrombus, infarct, abscess, gumma, tubercle, specific neuritis, multiple sclerosis, syringomyelia, polioencephalitis, meningitis, or ocular conditions. Here, disturbances in the visual mechanism, reflexly affect the vestibular apparatus.

If vertigo can be produced only by disease affecting the vestibular apparatus, then it follows that in order to diagnosticate the cause of vertigo, we must employ those tests which analyze the function of this apparatus. Such an examination consists of a search for any spontaneous vestibular phenomena which may be present, as well as an analysis of the responses to ear stimulation. Of the two methods, the latter is by far the more important. We start out with a fixed and definite idea that a normal internal ear with intact nerve pathways leading to normal nerve centers will invariably produce a rhythmic nystagmus and vertigo when a stimulus is applied. The nystagmus lasts twenty-six seconds in a certain direction when the individual is turned in a chair ten times, and the vertigo is also of twenty-six seconds' duration. Because of the vertigo there also appear past-pointing and falling. If, on the other hand, stimulation of the internal ear fails to produce one or all of the responses, it is positive evidence of an impairment along some point of this apparatus. A knowledge of the anatomy of this vestibular mechanism is, of course, indispensable, and one's ability to make a correct diagnosis as to the location of any lesion causing vertigo depends entirely as to how well he can visualize the anatomic pathways which constitute this vestibular apparatus as far as those pathways are known. The internal ear is the starting point for all impulses. Any form of stimulation applied to the internal ear, be it turning or douching, affects all nerve endings in like manner, and we think it highly improbable that there could be a lesion that would affect the hair cells concerned in the production of nystagmus without affecting also the hair cells concerned in the production of vertigo. If, therefore, stimulation of the internal ear fails to produce any response at all, or when all the responses are affected similarly, we conclude that the lesion is either in the labyrinth itself, or in the eighth nerve. If, on the other hand, stimulation of the internal ear produces one good reaction at the same time that the other responses are absent or markedly impaired, it is obvious that the internal ear itself and the eighth nerve are intact, and that the lesion is located centrally; that is, after the eighth nerve has divided into its component portions on entering the brain stem at the junction of the medulla oblongata and pons. If the nystagmus is impaired, the block is obviously in the vestibuloocular tracts; whereas, if it is the vertigo that is impaired, the block must be located along the vestibulocerebellocerebral tracts—those concerned with the production of vertigo. A knowledge of the course which all these tracts pursue within the brain substance enables one to locate accurately the site of the lesion responsible for the vertigo. However, it not infrequently happens that patients complaining of vertigo, when tested show normal reactions to ear stimulation. In these cases it is obvious that we are dealing with a condition where the vestibular apparatus is irritated at some point, and a search should be made for such a source of irritation. This may be a visual defect or a focal infection in the tonsils, teeth, stomach, kidneys, etc.

Therefore, just as we examine the urine in suspected cases of nephritis or diabetes, or as we have a Wassermann test made in suspected cases of syphilis, just so the ear tests enable us to analyze the apparatus responsible for dizziness. Instead of guessing, we then know the reason for the vertigo. Based on an experience of hundreds of examinations of pathologic cases in the Department of Neuro-Otology at the University of Pennsylvania, we do not hesitate to say that it is very rare that a "dizzy" case remains obscure after these ear tests, and in most instances the diagnosis becomes clear and simple. After these tests are made we are able to say, for example, that the patient is dizzy because of a toxic impairment of the labyrinth, a labyrinthitis, an organic lesion in some part of the vestibular tract, such as is produced, for example, by brain tumor, in internal hydrocephalus, brain abscess, or multiple sclerosis; or in the presence of normal reactions, we can state with assurance that the vertigo is purely functional or due to an evanescent toxin.

In conclusion, the following points may be emphasized:

1. There can be no vertigo unless there is a disturbance of the vestibular apparatus. When diseases in remote organs are

accompanied by vertigo, it is because those pathologic states somehow affect the vestibular apparatus.

2. Vertigo may be due to some simple irritation of the vestibular tracts. In such a case they are temporary and fleeting in character, leaving the apparatus itself intact, and the tests will therefore show normal responses.

3. There may be a lesion of the internal ear itself.

4. There may be a lesion situated within the brain along some pathway in association with the ear.

Disturbance of the vestibular apparatus with vertigo can be definitely analyzed and diagnosed by means of the ear tests.

XXVII.

CONCERNING THE USE OF THE ELECTRIC CAUTERY IN THE NOSE.

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An article in a recent number of the Annals of Otology, Rhinology and Laryngology condemns almost unreservedly the use of the electric cautery in the nose. Evidently, rhinologists are not all of the same mind on the subject.

When this therapeutic measure was first introduced, it naturally had as many advocates, both wise and foolish, as any other new therapeutic measure. After it had been used, and also abused, for a while, it naturally had as many adverse critics, both wise and foolish, as any other new therapeutic measure. Whether it is the sewing of a button on milady's dress or her jejunum, the pendulum will not be denied its swing. And there is a philosophy in the oscillation of the pendulum.

In contemplating the various movements of medical thought and action we cannot help being reminded of one of Pudd'nhead Wilson's maxims. To quote from Mark Twain: "We should be careful to get out of an experience only the wisdom that is in it—and stop there; lest we be like the cat that sits down on a hot stove lid. She will never sit down on a hot stove lid again—and that is well; but she will never sit down on a cold one any more."

Impulsiveness is frequently a characteristic of medical human nature. It is closely associated with enthusiasm. The one is to be condemned and the other commended. Too often, however, no distinction is observed, and doubt and hesitation exact their toll. Hence the swing of the pendulum, back and forth, and the truth, situated at either end of the arc, or at some point between, is discovered only by the philosophy embodied in the above quoted maxim. The lessons of history, of experience, too often pass unheeded.

Harm can come of almost any therapeutic measure, and the electric cautery is no exception. But it is hardly fair to deny, at the classic one fell swoop, the usefulness of the cautery when properly applied. For it is a most efficient weapon in the warfare against some very common nasal diseases.

There may be a dispute as to what constitutes the proper application of the cautery, but the conflicting opinions on the subject will eventually disclose the truth. A little more circumspection, a little more reflection, and a wide divergence may blend into an harmonious unanimity of opinion which cannot be used as a shuttlecock by the animadversively inclined.

The electric cautery is a most useful agent for combating certain nasal conditions, and when properly done and the wound subsequently treated, is attended by no harmful aftereffects. When too extensive an area is cauterized, and when subsequent treatment is curtailed or omitted, the stage may be set for pernicious sequelæ, such as persistent crusting and adhesions; and these constitute the chief objections to the abuse of the cautery, but not to its use. Temporary crusting always occurs, and continues for varying periods of time—a few weeks or a couple of months at the most. I cannot recall, at the present moment, a single case of permanent crusting. And in eliciting the symptoms of the patient in this particular, we should keep in mind the common crusting at Kiesselbach's spot, which may bias our conclusions if no note of it has been made previously. And here is the point. At Kiesselbach's spot the metamorphosed epithelium has two dimensions, length and breadth, while after a linear cauterization it has relatively but one dimension—length. The mucous membrane finds it easier to grow over a line than over an area. In many cases, after a comparatively short lapse of time, it is most difficult to determine, by the closest inspection, whether or not the cautery has been used. This applies particularly to those cases of simple vasomotor paresis without hypertrophy. Where hypertrophy or hyperplasia has been present, a linear depression or furrow may be visible; but as that is one of the results sought for in such cases, it is simply evidence that the cauterization has accomplished its purpose. And it is well within the limits of truth, supported by clinical experience, that linear cauterization practically removes the objection to crusting. When the nasal fossa is narrow, or when two opposite surfaces are cauterized at the same time, adhesions may result. In the former case, ordinary after-treatment, continued until the wound has healed, will serve as a preventive. And there is no reasonable excuse for simultaneous cauterization of opposing surfaces. Even when done accidentally, a little care and persistence in the after-treatment will obviate all difficulty.

The inflammatory reaction which follows the use of the cautery is occasionally pronounced, and in some cases—so it has been stated—has led to meningitis. Pneumonia, too, has followed the administration of ether as an anesthetic; but the careful anesthetist will be reasonably sure that no inflammation of the respiratory passages is present when the ether is administered. And the careful rhinologist will be reasonably sure that there is no active inflammation of the nasal mucosa present when he uses the cautery; and, in addition, he will cleanse the nasal fossæ before beginning the operation, and also avoid the olfactory region.

The subsequent inflammatory reaction cannot be seriously considered an argument against the use of the cautery. It is often mild; and even when severe, gives the patient no troublesome symptom excepting a temporary nasal obstruction, to which, indeed, he has already been subjected.

The alleged evil after-effects above mentioned—towit: persistent crusting, adhesions and excessive inflammatory reaction —are not inevitable sequelæ, and are likely due to errors of judgment and technic, or neglect of after-treatment.

It would serve no purpose to attempt an enumeration, to a body of rhinologists, of all the indications for the use of the electric cautery in the nose. But there are several types of cases which, in themselves, provide sufficient justification for its employment. First, those cases characterized by various degrees of vasomotor paresis of the turbinated bodies, especially the inferior, giving rise to alternate, intermittent, nocturnal or decubital nasal obstructions; second, cases of chronic rhinitis not secondary to some other nasal condition, and characterized by a thick mucous or mucopurulent discharge, with or without beginning hypertrophy; and third, cases of reflex irritation—e. g., asthma, the vasomotor rhinitis group, nocturnal enuresis, dysmenorrhea, etc.

Other methods of treatment are applicable, of course, in such cases. But there are times when other treatment fails, and it often happens that cauterization is attended by results which can only be described as brilliant.

XXVIII.

FACIAL PARALYSIS AS A COMPLICATION OF ACUTE OTITIS MEDIA.

By Edward Barton Jones, M. D.,

Washington, D. C.

Facial paralysis is not uncommon as a sequel of chronic suppuration in the ear, but its appearance in connection with acute otitis media does not seem to be of very frequent occurrence. On looking over the literature of the past ten years, I find that some of the books on otology do not mention it at all, and that some others give it scant notice. I found very few actual cases reported. There is a wide divergence of opinion as to its frequency and significance.

To explain the involvement of the facial nerve in an acute inflammation of the middle ear, reference must be made to their anatomic relationship. The fallopian canal is separated from the tympanum by a thin wall of bone, sometimes extremely thin. Often there is a dehiscence in this wall, usually situated just above the oval window, and the nerve, therefore, in such instances, lies behind the mucous membrane lining the cavity of the middle ear. There are other openings in the bony wall between the canal and the cavity, such as those for the passage of the chorda tympani nerve, the branch from the facial to the stapedius muscle, and the tympanic branch of the stylomastoid artery. It is believed that these offer channels for the spread of the inflammation. The impairment of the function of the nerve is brought about either by direct involvement, the inflammation extending to its sheath, or by pressure of exudation within its canal, or, in the opinion of some authors, by pressure from the tympanum. Ulceration and caries of the wall may take place, even in the acute stage.

The existence of the dehiscence is held by most writers to be the probable explanation of the way in which the facial nerve becomes affected, either as permitting pressure upon the nerve by the pent-up exudate in the tympanum, or as providing a favorable opening for the extension of the inflammation. It is difficult to see how an exudate in the tympanum pressing against an intact mucous membrane covering such a deficiency in the bone could exert sufficient force to produce a paralysis. It would be more reasonable, perhaps, to expect such an effect if the mucous membrane were intensely swollen and projected into the canal. If the paralysis were due solely to pressure upon the nerve from fluid in the tympanum, should it not immediately, or almost immediately, disappear upon relief of tension through the drumhead?

We are familiar with the fact that perforation of the drumhead may occur in a very early stage of otitis media and before the formation of pus. It is presumably a stronger wall, although of greater area, than the mucous membrane overlying the deficiency in the bone, and it may not be unreasonable to assume that the same process may take place in the latter with even greater rapidity and consequent exposure of the nerve to the action of the inflammatory exudate containing the exciting organisms.

Politzer says that whether a dehiscence in the fallopian canal favors the development of facial paralysis has not been proved; a fact speaking against it is that in caries and necrosis of the walls of the cavum tympani the facial nerve has sometimes been found lying free in the tympanic cavity completely surrounded by pus, without any symptoms of facial paralysis ever having been observed during the life of the patient. It might be inferred from this observation that to produce paralysis of the nerve (from middle ear disease) it must be subjected to pressure, such as could be exerted upon it while confined within its narrow channel.

The diagnosis is obvious. The paralysis may come on suddenly and be quite complete in a few hours, or it may be more gradual in its appearance, and certain muscles may be more affected than others. Severe pain in the ear and side of the face may precede its development; in other cases no pain is experienced, and the paralysis is ushered in by twitchings of the muscles. If the lesion of the nerve is limited in extent, its site in the canal can be determined, it is said, as follows:

If above the point where the chorda tympani takes its departure and below the branch to the stapedius muscle, the sense of taste in the anterior third of the tongue on the same side is disturbed. If above the twig to the stapedius there is, in addition, severe tinnitus and increased sensitiveness to low tones. If above the geniculate ganglion the sense of taste is not disturbed. There is alteration also in the secretion of saliva when the chorda tympani is involved.

The prognosis, as a rule, is good; a favorable outcome may be usually anticipated; however, recovery may be extremely slow, and in some instances permanent impairment may be the result.

The treatment of this condition is primarily that of the ear disease. If the middle ear inflammation receives proper attention the paralysis will usually disappear more or less promptly. Free drainage from the tympanum must be maintained; the membrane should be incised, if there is not already a perforation sufficiently large for escape of the exudate; if an existing perforation is too small for ready exit, it should be enlarged. In the absence of definite mastoid signs, operation does not seem to be indicated. Some authors recommend counterirritation over the mastoid and the internal administration of iodid of potassium. If the paralysis should persist in spite of improvement in the middle ear, electricity and massage should be employed.

This complication seems to be sufficiently rare to warrant the report of a case that came under my observation.

The patient was a boy, five years old. He had a history of glandular tuberculosis, particularly of the mesenteric glands, for two or more years, but seemingly had recovered from this disease. He had had chicken pox and a moderate influenza in the second week of February, 1915, but appeared to be entirely well during the next three weeks. His physician was again called to see him on March 8th, the boy having been awake all night with earache. On examination the ear appeared normal, and he was apparently well next day. The following day he again complained of pain; the membrane was pink, but not bulging, and its condition was the same on the 11th, and presented little change on the 12th, the paralysis beginning in the morning of that day. He had a temperature of 101°.

I first saw him on March 13th. The paralysis was almost complete. The membrane was red and bulging; on incision there was a serous bloody discharge, but no pus. A culture of the exudate showed a pure streptococcus of unusual variety. An autogenous vaccine was directed to be prepared.

March 17th. Paralysis very noticeably less. Temperature for several days has not been above 100°, and much of the time normal.

March 20th. Temperature, 102.6°; he felt sick.

March 21st. Paralysis practically gone. Temperature, normal. This day the vaccine was first ready for use and he was given a dose of five million.

March 23d. Cervical glands enlarged; no tenderness over mastoid.

March 24th. Temperature normal in morning; at noon a sudden pain in the ear, and temperature 101°. He received a dose of vaccine, seventy-five million. In the afternoon there was some swelling in front and below the ear; no appreciable tenderness over mastoid. Much redness and swelling in the upper half of the drum membrane, extending on to the meatal wall. Temperature 103° in the evening. Next day he felt very well; temperature near normal, swelling gone and membrane looking better. From this date the temperature was practically normal. He received four more injections of vaccine, at intervals of about three days, in increasing dosage, the last being five hundred million on April 8th, the date of last record of any discharge from the ear. The patient was then completely recovered.

During the course of the disease the meatus was kept clear by irrigation at suitable intervals. The ear drained well and the exudate maintained a serous character during two weeks, becoming slightly mucopurulent as it lessened in quantity, and ceasing in about four weeks from the date of the incision. The paralysis lasted about ten days. Whether or not the vaccine had any effect on the middle ear inflammation, it had no influence over the duration of the paralysis, as its use was not begun until the latter was ending.

XXIX.

PRIMARY JUGULAR THROMBOSIS DUE TO TONSIL INFECTION.*

By Charles Goodman, M. D.,

New York.

On December 8th a married woman, thirty-five years of age, was admitted to the Beth Israel Hospital. When five years of age she had had typhoid fever.

She had received treatment for follicular tonsillitis for ten days before her admission to the hospital. Five days previous she had had two or three chills, followed by fever and by profuse perspiration, accompanied with vomiting. A tender swelling was noticed on the left side of her neck, and she was referred to the hospital. Upon admission she had a temperature of 104°. She had a severe chill which was followed by a rise of temperature to 107.2°. The patient became delirious. The blood culture taken on the day of admission and again on the following day proved to be negative. Her blood count was 12,600, polynuclears eighty-four per cent. An examination made by Dr. Kopetsky of the ears, throat and accessory sinuses was negative.

On the evening of December 8th the patient presented an induration extending along the anterior border of the sternocleidomastoid on the left side. The patient appeared to be very ill and septic.

I confirmed the diagnosis made by Dr. Schneider, the house physician, of a probable septic thrombophlebitis; the chills, the hyperpyrexia and delirium indicating immediate operation. The pharynx was markedly congested, otherwise presented nothing abnormal.

The internal jugular vein was exposed by the usual incision along the anterior border of the sternocleidomastoid muscle. It was found surrounded by considerable infiltration of the tissues. The vein was exposed throughout its entire length and ligated near the clavicle. Thrombosis was situated at and included part of the facial tributary. The vein was resected from the clavicle below with a portion of the facial tributary to the jugular foramen. The greater portion of the wound was sutured and a drain was inserted in either end. The temperature gradually fell and on the fifth day was normal. The patient was out of bed on the fourteenth day.

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*This patient was presented before the Surgical Section of the New York Academy of Medicine, at a stated meeting held January 5, 1917.

During the operation the question arose as to the advisability of exposing the sinus, because I did not get blood readily with the aspirating needle introduced into the upper part of the jugular. I maintained that the danger of dissemination of infectious material throughout the body had been eliminated with the ligation of the jugular vein below the apparent thrombosis before the resection of the vein was undertaken. The exposure of the sinus was, therefore, not made, as the chiseling necessary for its exposure would have diminished the chances of the ultimate recovery of the patient.

Up to the present time there has been no indication for any further operative procedure.

The interesting features of this case are the direct infection of the jugular by the lymphatic route from the tonsils and the pharynx instead of the usual primary involvement of the sinus, and a secondary extension to the jugular complicating mastoiditis.

In recent literature, Long's case of sore throat with streptococcic invasion of the internal jugular vein and subsequent thrombi most nearly approaches this one.

Barnes, in a compilation of reports of systemic infections secondary to follicular tonsillitis involving the pharyngeal wall, mentions no direct infection of jugular vein as a complication.

We may conclude that the probable bacterial agent was streptococcus, since it is the most frequent factor in acute infectious processes in the mouth and throat (Braun) as well as in thrombosis.

It is interesting to quote here the explanation given by Libman and Celler of the negative blood cultures they obtained in a number of cases when sinus thrombosis of infectious origin was present: "We are inclined to believe that there may be early no bacteremia, that the bacteria may then be found perhaps in increasing number, and that it is possible for the bacteria to decrease in number and even to disappear before there has been any operative interference. It is also possible that in certain cases there is below the infected clot an obturating, noninfected clot which prevents the bacteria from entering the circulation. We can also imagine the possibility of there being an infected clot which completely closes the vessel. Another possible explanation is that in the course of the infection the blood may acquire a higher bactericidal power."

By analogy the same arguments might be applied to the negative blood cultures in our case of jugular thrombosis we have just reported.

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XXX.

TRANSILLUMINATION OF THE LARYNX AND UPPER TRACHEA.*

(FIRST COMMUNICATION.)

By Frank R. Spencer, A. B., M. D.,

Boulder.

In June, 1916, while the guest of Dr. Joseph C. Beck, at the North Chicago Hospital, I was invited to look into the larynx and trachea of a patient suspended with Lynch's suspension laryngoscope. The Beck light, on a stand, was at the patient's left shoulder, and the shade so placed that much of the light was thrown on the left side of the larynx and upper trachea. I chanced to view the interior of the larynx and trachea the instant before my head mirror could throw the light into the upper respiratory tract, and I had my attention immediately attracted by the readiness with which the interior was illuminated by the light on the stand. I called Dr. Beck's attention to this, and the word "transillumination" quickly suggested itself to his active mind. The thought that I had discovered something new occurred to me, but upon looking up the literature I found I was mistaken.

Whether this particular method of transillumination is of any real value, and whether this method of using it is original, I cannot say, but I think it is. However, transillumination of the larynx was first practiced by Czermak,[4] but was later perfected by Voltolini.[6] Freudenthal states in a letter that he has had experience with transillumination of the larynx, and that he was the first one in the United States to demonstrate the method. He has written articles upon this in German[2] and in English.[1] [3]

I shall present this method for what it is worth and let the future decide its real merit and permanent place in laryngology. However, I am afraid transillumination of the larynx and trachea has only very limited practical value when compared with transillumination of the maxillary sinuses, because it is so easy to obtain a good view of the interior of the former and so difficult to do so with the latter.

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*Read before the Western Section of the American Laryngological, Rhinolcgical and Otological Society, at Colorado Springs, March 17, 1917.

The following are the methods of applying transillumination to the larynx:

1. Voltolini[6] used a metal covered electric light for condensing the rays of light on the exterior of the larynx.

2. Freudenthal[2] improved upon Voltolini's model of laryngeal transilluminator and applied it either at the pomum Adami or near the cricoid cartilage, and introduced the heated laryngeal mirror into the dark pharynx so as to obtain a good view.

3. Cheatham has used a de Zeng transilluminator, but he does not say just how he applied this. Several other laryngologists have used this test, or method of examination, in different ways, judging from their replies to my circular letter of inquiry to all the fellows of this society.

4. The Beck light, on a stand, may be used to good advantage to light the larynx while the patient is suspended, as I mentioned in the first part of this paper. Whatever the method of applying this, the principle is the same.

Transillumination, even in normal patients, should be of value for inspection of the larynx and upper trachea. In cases of benign growths it should give a fairly definite idea of the amount of inflammatory thickening about the point of attachment. This will be of even greater value in cases of malignancy or suspected malignancy. The same may be said of tuberculous or syphilitic ulcers, lupus, chondritis, perichondritis, etc. Freudenthal in a personal communication relates the following: "I recollect one case which I saw, together with Dr. Lefferts and the late Dr. Asch. It was a man with terrific stridor. Both these colleagues had made the diagnosis of aneurism, while my diagnosis was carcinoma of the trachea. A dark shadow was plainly seen on transillumination. At the autopsy both conditions were found—i. e., a cancer, on top of which (or on the side, I do not recollect particulars) was an aneurism." As this was prior to the bronchoscopic and suspension laryngoscopic era in medicine, such a test was of more value then than now, other things being equal.

As the test can be made during either suspension or direct laryngoscopy, or by a modification of indirect laryngoscopy, it cannot be used as easily as transillumination of the maxillary sinuses, for instance. Direct or indirect inspection of the mucosa of the upper respiratory tract, by any of the standard methods, is of greater value than transillumination, especially since indirect inspection is comparatively easy. On the other hand, we should use in laryngology, as in the other specialties, all the tests at our disposal, if we cultivate accuracy in diagnosis. The average case may not and probably will not require it, but the difficult ones will, especially if direct or even indirect laryngoscopy is very difficult or for any reason impossible. I have in mind two patients with ankylosis of the mandible, as a part of a general joint infection due to tonsillitis, in whom it would be difficult or even impossible to examine the larynx except by transillumination. In fact, the ankylosis is so pronounced that it is difficult for them to masticate properly. While such cases are the exception to the rule, they do occur.

Schmidt,[7] however, has the following to say: "For laryngeal examinations, in which the transilluminator with a cover, as in frontal sinus examinations, is placed anteriorly on the larynx, I regard the method as without value. Besides, it is very difficult to place the mirror in the correct position in the dark pharynx. It is true that a transilluminated larynx gives a beautiful picture, but I have not obtained any practical results from the method."

Roth[11] does not favor transillumination of the larynx, as he believes the test is not practical.

Dr. H. Arrowsmith writes as follows in a letter: "Transillumination of the larynx was done, to a very limited extent, twenty-odd years ago. I tried it out casually at that time, but with others discarded the procedure as having no practical value."

The normal larynx shows shadows cast by the thicker portions, such as the true and false cords and the cricoid cartilage. The thyroid cartilage offers, of course, the greatest obstruction to the rays of light. The rings of the trachea cast very definite shadows.

The ventricle of the larynx, the cricothyroid membrane, and the membrane extending between each two adjacent tracheal rings admit more light than the thicker structures, hence, at any of these points inflammatory thickenings, benign or malignant growths, will show most distinctly on account of the shadows which they cast. The same may be said of the membrane extending from the lower border of the cricoid to the first ring of the trachea.

During the past fifteen months I have had under observation a papilloma of the larynx in a man, aged forty-one years. This growth has returned twice following removal by suspension laryngoscopy. The papilloma originally sprang from the left ventricle, but later seemed to be attached to the inferior surface of the false cord, too. This casts a distinct shadow by transillumination, during suspension laryngoscopy, and shows well the inflammatory thickening at the point of attachment. While the pathologist reported a suspicion of malignancy from the second papilloma removed, I do not believe it is malignant, but the shadow cast is due purely to the thickened tissue.

Cases examined with the bronchoscope or esophagoscope for a foreign body or a stricture, it is needless to say, show only the normal shadows. Imperatori states in a letter: "I have noted the light passing through the cricothyroid membrane while suspending the patient and with the long lighting attachment within the larynx." Since I have not had an opportunity to use this in a single case of malignancy, I cannot speak from experience of its value here.

J. Solis-Cohen in a personal communication writes as follows: "I have tried transillumination of the larynx and trachea, but got very little satisfaction from it. The parts looked like translucent candy, and it was not possible to outline growths and structure, so I gave it up. I do not think I ever wrote upon the subject."

Schech[5] makes the following interesting statement: "The transillumination of the larynx, first practiced by Czermak, and later being perfected by Voltolini, has no diagnostic value."

Dr. Frank E. Miller writes as follows: "I have had at least fifteen years' experience with transillumination of the larynx and trachea. In each I find it especially helpful, where there are gouty deposits and tuberculous combinations. It has proved extremely satisfactory and has been particularly helpful in diagnosing cancer cases, by exclusive processes. At the present time I have three cases of gouty deposits of the thyroid and arytenoid where the above had not been employed, causing improper recognition and tracheotomy. In a number of cases where this has shown simply a shadow overlying the thyroid gland, the penumbra has resulted in serious errors of diagnosis."

Frankel[8] has referred to transillumination of the larynx in Simon's handbook of laryngology, but mentions this very briefly.

Dr. Norval H. Pierce states: "I have had some experience with transillumination of the larynx and trachea, and now have a paper in preparation on the subject. This procedure has been used before, as a paper was read on the subject before the Belgian Laryngological Association."

A. L. Benedict[9] has suggested the use of transillumination of the upper respiratory tract.

Wm. Cheatham, in reply to my letter of inquiry, states: "A few years ago I became interested in laryngeal transillumination and used de Zeng's frontal sinus transilluminator. I found the study very interesting."

Dr. Lee M. Hurd writes as follows: "Four years ago, while observing a bronchoscopic tube being passed in a darkened room, I noticed the translucency of the light on the neck and chest wall as the tube was passed, and later reversed the idea of light outside, using mirror to examine larynx and trachea. I have used the method for observing the contrast and to outline growths in the larynx. Lennox Brown used a similar method thirty years ago."

To quote further from Freudenthal:[1] "Now, to get a view of the interior of the larynx, we apply this lamp to the external surface of the neck, either at the pomum Adami or near the cricoid cartilage, and introduce the heated laryngoscopic mirror into the dark pharynx, and thus we obtain a splendid view of it. But this view is quite different from that ordinarily seen. The first thing that strikes the observer is the disappearance of different colors. The whole larynx shows a reddish tint of varying intensity. When we place the lamp at the level of the incisura thyroidea, then the vocal bands and all parts above them appear of a beautiful red color (of course the epiglottis is dark). But when we place the lamp near the cricoid cartilage, then we get a better survey of the whole subglottic region down to the bifurcation of the trachea, and this view is often more satisfactory than that obtained by the common method. But it must be observed that we have to become accustomed to this peculiar view of the larynx, for, it may be said, we see mostly in the negative. What, in ordinary illumination, impressed us as a thickening or an enlargement of a solid mass, in transillumination strikes us as a dark object. The rays of light cannot penetrate, and we infer that a solid mass, or a mass subject to the same optical laws, must intervene. We will, therefore, be able to define more precisely the contour of a tumor, because we see how the mass differentiates itself sharply by its dark outlines from the other parts in view. Only a few days ago I saw a patient of Dr. L. Weiss, who had a tumor on the posterior wall of the larynx, extending between the two cartilages from the left arytenoid downward. We can see the circumference of it by means of transillumination better than by the ordinary method. I will have the pleasure of showing you the patient, and will be very glad to have the benefit of your opinion upon the case."

"Furthermore, we will be enabled to differentiate, in a certain case, between a solid tumor and a cyst, and this point seems to me to be of sufficient importance to give the method a fair trial. A few weeks ago I operated on a man who, a few minutes afterward, happened to be seen by Dr. Hoch, of Philadelphia. The patient had a tumor, the greatest part of which was below the glottis. It was attached to the right vocal band at the front angle, and extended over a little to the left vocal band. By means of transillumination I could easily determine that it was a solid tumor, not a cyst, and could gauge the exact size of it. It is true that you could have excluded a cyst in this case even without transillumination, but there are cases where this is not so easily done, and for these I think transillumination is also of value."

"In this man, by the way, we could see, the day after the operation, that the edematous vocal band was much more translucent than before the edema had set in, and this brings us to the other class of cases, where we have a greater penetration of light than normally. This, however, can only be seen where loss of substance has taken place, as in ulcerative processes or in other pathologic conditions which admit a greater penetration of light. When Gottstein[10] says, 'the reddened vocal band seems just as translucent as the normal,' he is correct; but you cannot draw from that a conclusion as to the usefulness of this method generally. Certainly we cannot recognize these changes, but there are others which we can see so much the easier. So, for example, an edematous vocal band is differently translucent than an infiltrated one. The first is of a strong, red light—i. e., much redder than the normal—the last is much darker. This is a differentiation which is interesting enough to us."

While I have used transillumination in a few cases, my experience is too limited to warrant many positive conclusions, so I will, therefore, submit this test or method of examination for your approval in this "first communication," but I shall report more in detail later.

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XXXI.

THE RELATIONS OF THE SPHENOID SINUS TO THE SEMILUNAR (GASSERIAN) GANGLION AND THEIR POSSIBLE CLINICAL IMPORTANCE.*

By Greenfield Sluder, M. D.,

St. Louis.

The semilunar ganglion is at present thought of as so far removed from the sphenoidal sinus that they have not been associated in the minds of the anatomists or clinicians. The internal carotid artery usually rises on the lateral aspect of the body of the sphenoid, and the semilunar ganglion is usually lateral and posterior to the ascending artery. This, however, is not always true. The position of the artery seems to have a large part in determining these relations.

I present drawings of two specimens showing an intimate association of the sphenoid sinus to the semilunar ganglion, or parts of it. Figure 1 shows dissection of the cavernous sinus, semilunar ganglion and sphenoidal fissure, viewed from above. The sphenoid sinus, lying beenath these structures, is shown in dotted line. The thickness of bone separating the cavity of the sphenoid from these structures is egg shell thin. The internal two-thirds of the ganglion are exposed to the uppermost part of the sphenoid cell, and the external third is exposed at a little greater depth as the nerve tissues approach the foramen ovale. The mandibular nerve in the foramen ovale is exposed for ten millimeters to an egg shell bone separation from the sphenoid cell.

In the sphenoidal fissure the oculomotor, abducens and ophthalmic are exposed to an egg shell separation from the sphenoid cell. The trochlearis alone is not in this contact, because it lies on top of the oculomotor.

Figure 2 shows a sagittal section through the oculomotor nerve within the cavernous sinus. The specimen is viewed from within outward. In this specimen the ganglion is exposed on its anterior limit for ten millimeters to an egg shell thin bone separation from the sphenoid sinus at the origin of the ophthalmic and maxillary divisions. The sphenoid cell measured 1.50 centimeters sagittal, 4.50 centimeters vertical, 5.50 centimeters transversely.

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*Read before the meeting of the American Laryngological Association, May, 1916.

In 1912[1] I proved the permeability of the sphenoid sinus wall to small amounts of cocain. Following this observation, with its lesson, into these anatomical associations seems to me to offer an explanation of the herpes which develops in the wake of sphenoidal infections, or which arise in some patients from ordinary coryzas (irritation of the sensory ganglion cells being necessary for the development of herpes), and to explain why semilunar ganglion neuralgias and tic douloureaux of sphenoidal origin[2] sometimes recover as a more or less acute or subacute lesion, and at other times requires a ganglion removal or a posterior root section—i. e., sometimes the sphenoid lesion can be controlled and at other times it cannot.

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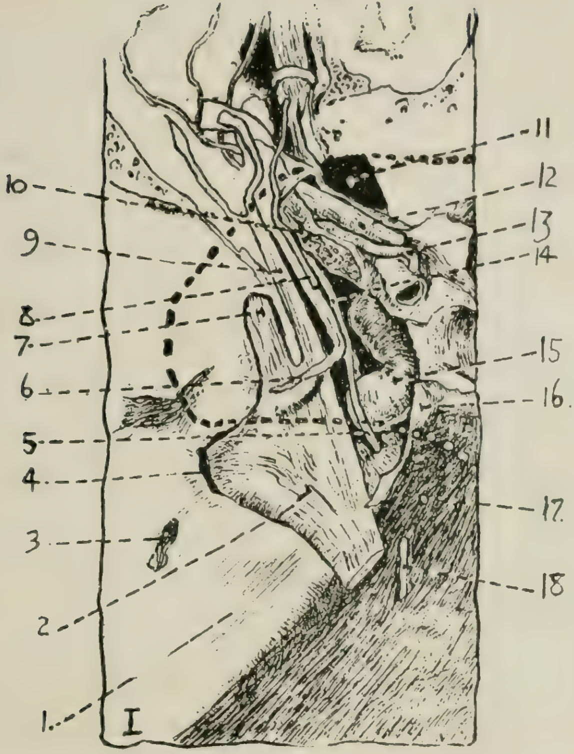

FIGURE 1.

Top view of left sphenoidal district, dissected. 1. Petrous part of temporal bone. 2. Semilunar (Gasserian) ganglion. 3. Foramen spinosum. 4. Foramen ovale with mandibular nerve in it. Inside the sphenoid cell the foramen ovale containing the mandibular nerve marked as a canal five millimeters long, separated in a semicircular exposure from the sphenoid cell by bone one millimeter thick. 5. Abducent nerve. 6. Oculomotor nerve. 7. Maxillary nerve entering foramen rotundum. 8. Trochlear nerve. 9. Ophthalmic nerve. 10. Inner limit of sphenoidal fissure. 11. Opening dissected into sphenoidal sinus. 12. Optic nerve. 13. Ophthalmic artery. 14. Abducent nerve. 15. Internal carotid artery. 16. Posterior clinoid process. 17. Clivus of Blumenbach. 18. Abducent nerve. Dotted line shows outline of sphenoidal sinus. It is separated from the overlying structures by a wall of bone, egg-shell thin, including those in the sphenoidal fissure. The inner two-thirds of the semilunar ganglion for almost one-half its length are also included in this close associati.on.

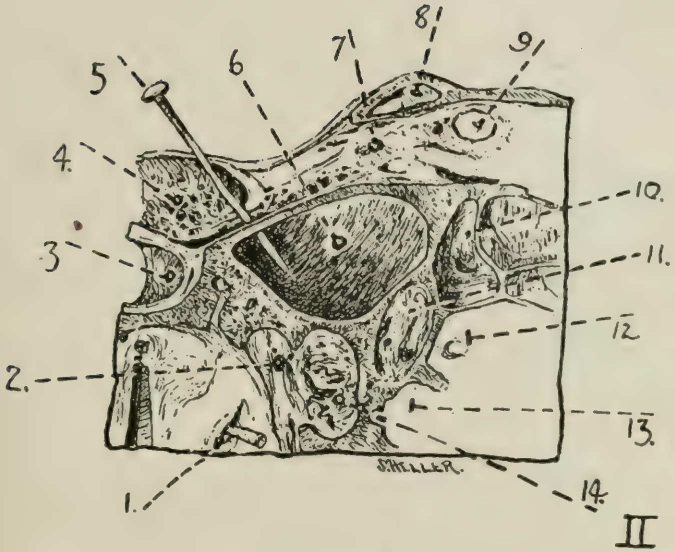

FIGURE 2.

Shows a sagittal section of left side through the ophthalmic nerve. 1. Eustachian tube with bristle in its lumen. 2. Tensor palati muscle. 3. Part of internal carotid artery. 4. Semilunar ganglion in Meckel's cave. 5. Pin passed through wall of sphenoid. 6. Sphenoidal sinus. 7. Ophthalmic nerve. 8. Lesser wing of sphenoid. 9. Optic nerve. 10. Postethmoidal cell. 11. Pterygomaxillary fossa. 12. Opening dissected into maxillary antrum. 13. Wall of nose. 14. Internal pterygoid muscle.

XXXII.

IS A MODIFIED RADICAL, OR HEATH OPERATION, A JUSTIFIABLE PROCEDURE?

By A. Spencer Kaufman, M. D.,

Philadelphia.

The modified radical, meatotympanic, or Heath operation, like many other surgical procedures advocated, is very much abused, both in its application and by its critics. Many times the one who recommends these methods is carried away by his own enthusiasm, and advises too free use of his operation, and when others fail to get results, there is general condemnation. Then, again, the users often fail to follow indications and to properly select their cases, and again the operation falls into disuse, regardless of the good there may be in it. I believe the Heath operation suffers for the first reason.

The modified radical mastoid operation was first advised by Körner in 1899, and later by Heath, Bryant and Ballenger. Jansen also claims to have described this operation in 1893.

Briefly stated, the operation consists in doing a simple mastoid or Schwartze operation, and the removing of the posterior bony wall of the canal to within a quarter of an inch of the annulus tympanicus, dividing the cartilaginous canal into flaps, and closing the posterior wound entirely. The contents of the middle ear are not disturbed in any way, and all postoperative treatment is carried out through the external auditory meatus. Every bit of mucous membrane must be removed from the antrum. In other words, you stop just short of doing a complete Schwartze-Stacke operation.

It has for its object the cure of suppuration, the repair of the drum membrane, and the restoration of hearing.

It has been asserted by some operators that a case requiring a radical operation will not get well with a Heath; and a case that does get well with a Heath would have made equally good recovery with a simple; and this is true to a certain extent. But there is a certain class of cases to which this operation is so admirably adapted that I believe it has a real place in otitic surgery—not to supplant either the radical or the simple, but to take care of a group of cases in which it is indicated.

In a large measure, however, it can be used to advantage in a great many cases in which we have heretofore felt that an ossiculectomy would serve.

The cases of chronic suppurative otitis media which actually indicate the employment of intratympanic surgery alone are exceedingly rare; indeed, I had despaired of finding such a case until the past few days, when a case turned up with the qualifications for an ossiculectomy. This operation will answer in that very limited class of cases in which the suppuration is limited entirely to the tympanic cavity, the discharge being caused by granulations and necrosis in the cavity and of the ossicles. Here, as in a constantly increasing number of conditions, the X-ray plays an important part. Given a case of chronic suppuration of the middle ear, the X-ray examination showing the presence of cells in the mastoid process not filled with pus, not yielding to local treatment, the operation for removal of ossicles is indicated; and I submit an X-ray plate taken by Dr. Willis F. Manges, revealing just such a condition, and another plate, for comparison, showing an eburnated mastoid, in which we would expect to find an antrum necrosis, and in which the removal of ossicles would be of little, if any, service.

In cleaning out the tympanic cavity in a radical mastoid operation the malleus and incus are, in the majority of instances, found either whole or with very little evidence of necrosis. This is true, even though the otorrhea has continued over a long period, and the examination through the canal shows destruction of the entire tympanic membrane, and the ossicles are not seen in the tympanic cavity proper. This fact leads me to believe in the futility of an ossiculectomy, and the possibility of the repair of the drum membrane and the restoration of hearing when the ossicles are in almost normal position and only a part of the tympanic membrane destroyed.

In cases of chronic suppuration, with the destruction of the greater part or all of the membrane, and the ossicles missing, or drawn up into the attic, and a roentgenograph showing either necrosis (pus) or a dense mastoid, but one operation is indicated, and that is the radical, or Schwartze-Stacke. As one of the objects of the Heath operation is the regeneration of the drum membrane, we certainly could not expect to have that result brought about when the ossicles have been drawn into the attic and could not again be gotten into normal position. Indeed, one would feel that if the membrane did reform under these circumstances, it would not act upon the ossicular chain, and impair rather than improve the hearing, thereby defeating the efforts for an improvement in function; and if the drum is not restored, we have only avoided the danger of injuring the facial nerve, and face the possibility of another operation.

For cases of acute mastoiditis not yielding to nonsurgical measures, the simple mastoid operation is unquestionably the one of choice. As the early and free incision of the tympanic membrane prevents all but a very small percentage of acute otitis cases from going to the point where a simple Schwartze operation is necessary, so in turn the simple operation, done early enough, will avoid the necessity for either a Heath or a radical operation.

If it is not intended to supplant either the simple or radical operation, where, then, are we justified in using it? There is a certain number of acute cases with which we come in contact that have gone too far to yield to a simple operation, and, on the other hand, do not demand a radical; or cases which would yield to a simple operation only after an abnormally long period of drainage and dressing. Prolonging the period of convalescence in this way frequently interferes with a patient attending to his usual work, on account of his wearing the bandage when he is physically fit to earn his livelihood. During the performance of a simple mastoid operation the bony canal is occasionally found to be very necrotic. To remove all of the necrotic bone would subject the patient to the danger of a collapsed canal, and to perform a radical operation under these circumstances would result in loss of hearing; whereas, if the tympanic ring is left intact and the contents of the middle ear cavity undisturbed, the result on hearing will be the same as in a simple operation. Then we find chronic cases with the disease confined to the antrum and mastoid proper, and the ossicles in place and a goodly portion of the membrane remaining. The membrane, in these cases, after a Heath operation, will reform, and the hearing be much improved. The typical case for a Heath operation is one of this type. The patient complains of a discharge having continued for some period, and examination reveals a large perforation of the tympanic membrane and the ossicles plainly visible. The X-ray will show either necrosis or eburnation of the mastoid, with necrosis of the antrum.

To illustrate, I wish to report a case typical in its indications and consequently satisfactory in its results.

Mr. C. S., aged about forty years, in September, 1912, felt a sudden fullness in his right ear, examination of which, the following day, showed a very red and bulging membrane. This was incised and pus evacuated from the middle ear. Three weeks later he had a similar occurrence in the left ear. The discharge from both ears continued and resisted all efforts to control it, and in the meantime he was becoming progressively deaf. I advised a simple mastoid operation, which was refused, and he went to another physician. In February, 1913, he came back to me, the discharge still continuing, and a destruction of the lower third of the membrane on both sides. His hearing at this time was absolutely nil, and it was necessary to write anything one wished to communicate to him. A Heath operation was advised and consented to. His physical condition not being good, the two sides were operated upon a week apart, in February, 1913. The patient went on to an uninterrupted convalescence, the drums regenerated, and his hearing improved up to the point where it is now, and he has no difficulty in getting along with ordinary conversation. There was a small area in each mastoid where dermatization was delayed for some time, but this did not interfere with his hearing or middle ear.

CONCLUSIONS.

It is a justifiable operation in—

First—Chronic mastoiditis where there is only partial destruction of the tympanic membrane and the ossicles are in position.

Second—In cases of acute mastoiditis with an unusual amount of destruction of the tympanic membrane and loss of hearing.

Third—In cases of acute mastoiditis with extensive necrosis of the bony portion of the external auditory canal.

It should never be attempted where there are cholesteatomatous masses found in the antrum, which indicate the radical operation; and it should never be done until all nonsurgical methods, including vaccines, have been tried.

XXXIII.

A CASE OF ACUTE MASTOIDITIS WITH AN ENORMOUS LEUCOCYTE COUNT—BOTH MASTOIDS OPENED—RECOVERY.

By George F. Keiper, M. D.,

La Fayette, Ind.

On January 4, 1916, through the courtesy of Dr. E. T. Mitchell, of Romney, Indiana, I saw at my office Alva B. S., aged four years, of Clarks Hill, Indiana. The left mastoid was the seat of acute inflammation, with suspicious symptoms in the right mastoid.

Four weeks before, he had an attack of tonsillitis, which eventuated in a case of purulent bilateral otitis media. Tenderness of both mastoids was rather marked. Under the ordinary treatment the inflammation in the right mastoid seemed to clear, until at the time of his visit to me, as above, it did not seem at all tender. But not so with the left mastoid. It continued to grow worse, and hence he was brought by his doctor to me. The left ear stood out from the head markedly. Tenderness over the mastoid was exquisite. The posterior superior wall of the external auditory canal sagged markedly, hiding the upper portion of the membrana tympani. The drum was perforated irregularly in the posterior inferior quadrant. From the perforation pus issued under pressure. A smear of it showed a mixed infection with a large number of streptococci present.

The right mastoid did not seem tender at all, neither was there any sagging of the external auditory canal present in the right ear. Few streptococci were found in the discharge of pus from the ear. The membrana tympani was ruptured irregularly in the posterior inferior quadrant.

The boy was sent to St. Elizabeth Hospital for blood counts, X-ray plates, and urinary analysis. The urine was normal.

The blood count was as follows: Hemoglobin, 90 per cent; leucocytes, 60,090; polymorphonuclears, 91 per cent; small lymphocytes, 8 per cent; large lymphocytes, 1 per cent; mast cells, none; eosinophiles, none.

Astonished at this enormous leucocyte count, I had another one made the same day, which verified in all respects the first one. In fact, it is the largest leucocyte count that I have ever encountered in mastoiditis. Search of the literature fails to reveal to me a parallel in this disease.

His temperature on admission to the hospital at 4 p. m. was 100.2°, and his pulse was 134.

I advised opening the left mastoid as soon as possible. X-ray plates showed it cloudy; the right one seemed normal.. This consent being given, we performed a simple mastoid operation the next morning. The cortex was thin, and pus was soon reached directly under it. All diseased bone was chiseled and curetted out clean. The wound was sewed up above, packed lightly and bandaged, and the patient returned to bed.

On January 6th the leucocyte count showed a decided drop to 16,500 per cubic millimeter. The polymorphonuclears dropped to 80 per cent, the small lymphocytes increased to 18 per cent, and the large ones to 3 per cent. By January 8th the leucocyte count had further dropped to 11,000 cubic millimeter. The polymorphonuclears had dropped to 72 per cent, the small lymphocytes had increased to 22 per cent, and the large ones to 6 per cent.

After the operation on January 5th the temperature at two o'clock was 99°, the pulse 144. By seven o'clock the temperature was 102° and the pulse 162. At eleven o'clock that night the temperature was 100°, but the pulse remained high, 160.

The temperature dropped on the 6th to 98°, with a pulse of 130. On the 7th it had dropped to 97°, with a pulse of 118. On the 8th the highest temperature recorded was 99.2°, with a pulse of 124. On the 9th it was 99°, with a pulse of 120. On the 10th the highest temperature was 98°, and the pulse was 116. The 11th, 12th, 13th and 14th showed like records. But the leucocyte count had increased, as shown by the chart of the blood count submitted herewith. It has been my own observation that when the pulse runs high out of ratio to the temperature, further trouble is brewing, and frequent blood counts are indicated to warn us of further infection.

On January 5th the temperature was 100.8° by axilla, and the pulse was 148. At five o'clock it was 101.4°, and the pulse 140, and so it continued until the 16th, when a marked swelling appeared behind the right ear over the mastoid process, with increased tenderness and bulging of the posterior wall of the external auditory meatus. The blood count showed 15,300 leucocytes per cubic millimeter, and the percentage of polymorphonuclears was 80. The pulse was 130 and the temperature was 100°. I advised opening the right mastoid, which was accomplished by a simple operation at eleven o'clock the same morning.

The first incision through the skin brought pus in abundance. In uncovering the mastoid process we found a perforation in the bone about five millimeters in diameter. With chisel, curette and burr we cleaned it out. In doing so we found the tegmen eroded and necrotic, and in removing this we uncovered the dura mater, which seemed normal in all respects. The wound was dressed as in the first instance and the patient was returned to bed. By two o'clock the temperature had dropped to 98°, with a pulse of 130.

As will be noted by a study of the accompanying temperature and pulse chart, the former ranged rather high until January 21st, when the temperature rose to 104.6°, at 7:30 p. m. At this time tincture of aconite in drop doses every hour was given until the temperature was reduced. No symptoms of meningitis or lateral sinus obstruction at any time were apparent. By January 22nd the temperature had fallen to normal, with a pulse corresponding to the temperature. Barring a few exacerbations, controlled by tincture of aconite, it continued to remain normal, with a normal pulse, until dismissed from the hospital, February 8th. The blood count in the meantime had become normal.

In addition to the aconite the internal treatment consisted in giving three grains of hexamethylemin tetramin every four hours during his waking hours during his stay in the hospital. Paregoric in ten drop doses was used to control restlessness, which was quite marked at times, especially at night. Quinin bisulphate in oleic acid was occasionally rubbed into the abdomen for the control of the temperature, though it was not as efficient as the tincture of aconite. The bowels were kept well open at all times, and high rectal injections were used to assist the medicinal treatment. Twice during the progress of the treatment the kidneys refused to secrete normally. Basham's mixture (liquor ammoniæ acetatis, fl. oz. vj; acid acetic, oz. iij; tinct. ferri chlor., fl. oz. v.; alcoholis, oz. ij; syrup, fl. oz. iv; aquæ, fl. oz. iv) was given with good effect. Hot stupes were also used over the kidneys. On the 17th and 20th a mixed stock vaccine was given hypodermically. Its value was very doubtful.

The most unusual feature of this case was the enormous leucocyte count of over 60,000 white cells per cubic millimeter. For a four-year-old boy, with comparatively small mastoid antra, it seems almost inconceivable to say the least. But the repeated blood counts, made the same day, leave no doubt as to the correctness of the observation.

As already intimated, the study of the temperature and pulse chart is fraught with interest.

Another interesting fact is that with so grave an infection the patient recovered, showing the great resistance possessed by this lad.

Were I to do this work over again, I would most certainly open both mastoids at the same time, in spite of negative X-ray diagnosis.

THE LEUCOCYTE COUNTS:

	Jan. 4	Jan. 6	Jan. 8	Jan. 10	Jan. 11	Jan. 12	Jan. 13	Jan. 16	Jan. 17
Leucocytes	60,090	16,500	11,000	12,400	11,600	15,200	15,600	15,300	15,000
Polymorphonuclears	91%	80%	72%	76%	84%	84%	84%	80%	77%
Small lymphocytes	8%	18%	22%	19%	12%	9%	11%	17%	19%
Large lymphocytes	1%	3%	6%	5%	4%	7%	5%	3%	4%
Mast cells	0%	0%	0%	0%	0%	0%	0%	0%	0%
Eosinophiles	0%	0%	0%	0%	0%	0%	0%	0%	0%

XXXIV.

AN OBSCURE CASE OF SIGMOIDAL SINUS THROMBOSIS.

By T. H. Odeneal, M. D.,

Beverly, Mass.

Patient, seen day after admission to the hospital, complained of pain in the mastoid region, not severe, tender about the tip; ear discharged a thick creamy pus. Temperature normal in morning, elevated to 99° in the evening. Patient was a thick set, robust German, and did not understand why I wished to operate, as he felt in such perfect health. Duration of symptoms one week.

Believing I had a simple mastoiditis alone to cope with, the mastoid was opened and a fistula found leading from beneath the cortex to the sigmoidal sinus, the bony covering of the sinus being entirely absent and the sinus found filled with a clot from the knee to the bulb. The jugular was ligated and the clot turned out, the patient making an uneventful recovery.

This case is interesting because of the lack of diagnostic symptoms pointing to a sinus infection.

I am almost led to believe that the thrombosis was an earlier affection and the mastoiditis merely a recrudescence of a previous one, which would account for the lack of any more definite symptoms.

Another case I have recently operated upon was seen with a large swelling behind the ear, reaching from the tip of the mastoid into the temporal fossa, which fluctuated on palpation. The temperature was normal, occasionally reaching 99° in the evening, and there was absolutely no pain or tenderness whatever.

A diagnosis of postauricular abscess of unknown origin was made, without an involvement of the mastoid. About two ounces of a creamy pus escaped on making an incision in the fluctuating mass. About two centimeters from the tip two small fistulæ were discovered which led on probing deep into the mastoid cells backward toward the sinus. On following up these two fistulæ with the chisel they were found to end at the membranous wall of the sinus, the bony covering having disappeared for a distance of two centimeters or so.

No granulations being present on the sinus, and the pulsation apparently normal, the sinus was not opened. The remainder of the mastoid was very much sclerosed. Patient is now making a rapid recovery, temperature remaining normal since operation, six weeks ago.

These two cases are very good examples of how utterly impossible it is to judge of the extent of a mastoiditis from a symptomatic standpoint, and how necessary it is to be on our guard constantly.

XXXV.

COMPLICATED GRADENIGO'S SYNDROME—OPERATION—RECOVERY.

By Adair-Dighton, M. D.,

Liverpool.

The following case seems, on account of the extreme diversity of the symptoms, to be worthy of report:

John B., aged sixteen years. Admitted to the Eye and Ear Infirmary on August 9, 1916, with a history that he had been taken suddenly ill four days previously.

On examination, patient was obviously very bad. Was reported by own doctor to have had a rigor; was semiconscious; complained of intense frontal pain, tenderness over left mastoid, profuse discharge from left ear. Temperature, 104°; pulse, 104; respirations, 28.

Dr. Edgar Stevenson examined his eyes, but reported both discs normal. Marked nystagmus of left eye to right side. Left ear not totally deaf.

On following morning he was moved to the operating theatre, and when on the table it was noticed that he had developed a marked strabismus in the right eye to the left, which was easily proved to be due to a complete paralysis of the right external rectus muscle. This had not been present on the previous evening. The right ear was normal, but there was a history of a "one-time" suppuration in it. Under chloroform anesthesia I operated and performed a very complete radical mastoid. The antrum was full of semifluid pus under pressure, and, as might be expected from symptoms, there was a distinct fistula in the external horizontal canal. Dura and sinus were purposely exposed and found healthy.

On following day the temperature had dropped to normal, where it remained throughout convalescence. Pulse, 98. Nystagmus and squint, no change.

The convalescence was normal, the nystagmus ceasing three weeks after operation, and the strabismus gradually clearing until on the 16th of September patient was discharged with the external rectus acting perfectly and a dry left ear. He had no refractive error.

Now this case is of interest:

1. On account of the alarming character and diversity of the symptoms.
2. The paralysis of the opposite external rectus.
3. The sudden cessation, after operation, of the alarming symptoms.

First, the symptoms of the typical Gradenigo syndrome have been so well described in the ANNALS by Hays, Perkins, Myers and others, that no addition of mine need be added, but throughout the whole of the literature on the subject I can find no mention of a case in which the abducens paralysis affected the opposite eye, and am at a loss to account for it in any way except the possibility of a reflex action lighting up an old-standing focus of disease in the right ear, and so causing a neuritis of the sixth nerve on the right side. Even with this as a possible explanation, I cannot see that it accounts for the absolute lack of symptoms in that ear, as there was never any sign of discharge. I have thought of the possibility that the right eye was a squinting eye of old standing, but failed to get any history of it, and there was no error of refraction.

SOCIETY PROCEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of January 12, 1917.

Paper: The Practical Application of Recent Work on the Internal Ear.*

By Isaac H. Jones, M. D.,

Philadelphia.

(By invitation.)

Paper: Vertigo: Its Causes and Methods of Diagnosis by Ear Tests†

By Lewis Fisher, M. D.,

Philadelphia.

(By invitation.)

DISCUSSION OF DR. JONES' PAPER.

Dr. B. Alex. Randall (Philadelphia) said: Supporting the claims of his colleagues, he would add little to what they had presented, but rather enforce some points perhaps too lightly touched upon; for he adopted the maxim of Michael Angelo: "Perfection is made of trifles: and perfection is no trifle." The chair which Dr. Jones has designed and illustrated is a considerable improvement upon Bárány's, still more upon the substitute shown in some of the pictures, since it places and holds the head in the exact plane desired, as near as possible to the axis of rotation. While nystagmus may be well evoked with approximate arrest of the turning, it is to be noted that for past-pointing the chair is always locked—best by a pedal. Poor appreciation of the exact planes of the semicircular canals has given rise to much faulty testing; the external canal, which many of us incline to call the horizontal, is thirty degrees from the horizontal, as shown; while the vertical canals are each some forty-five degrees away from either the sagittal or the frontal plane, by which some would name them—facts of fundamental importance. Then, just as we must abandon the misleading names like "crista acoustica" and "macula acoustica" for structures devoid of acoustic function, so must we cease to speak of the "static labyrinth" when dealing with the canal apparatus concerned in the perceiving and analyzing of motion. This is kinetic, not static.

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*See page 436. †See page 511.

The primordial aural organs, with their otoliths, are mainly static; although they may show not only the direction of gravitation, but somewhat appreciate motion and their hair cells recognize sonorous as well as other vibrations. Only in the more mobile animals, as the fish, does a semicircular canal apparatus appear, with a nonotolithic end organ in the ampullæ capable of recognizing and analyzing endolymph flow. The sacs of the vestibule have even in man their otolith organs, and it is of interest to note how the two "maculæ" are set at right angles to each other the better to fulfill their static function.

Man, trying to improve upon nature, has copied his camera from the eye, and in some essentials his telephone from the ear; and the aviator, in designing a "stabilizer," can probably do no better than follow the suggestion of the semicircular canals in their three planes at right angles to each other. For we may take it as the essence of all these phenomena—separate and independent, as Dr. Jones has told you the nystagmic are from the vertiginous symptoms—all are efforts, though perhaps perverted, to stabilize the sensorium. The patient feels as if failing in one direction, overcorrects, and so falls to the opposite side. He points to an object of known position; but while his hand is moving away and back, he changes his aim in exact proportion and direction as he subconsciously conceives his body to have turned from its primary position. Thus he gives a quantitative as well as qualitative indication of the stimulus of his kinetic apparatus. This is exactly the use which Graefe made of this test, using it to demonstrate the extra innervation given to paretic eye muscle.

This quantitative value requires that the tests be made with minute accuracy, unmodified by extraneous and perhaps calculated corrections; and shall be as rigidly recorded. To this end the charting which shall place this mass of facts clearly, almost graphically, before the examiner so that at a glance he can see all the essentials and note any flaws or perversions of the findings, is of prime importance to the securing of all the facts and to the analyzing of their meaning. Yet we have seen men try to carry in their heads the results of their inadequate testing, summing up as they went along to a futile conclusion; and then ready to condemn the whole investigation as worth no more (in this and presumably every other case) than the primary observation that the patient walked like a cerebellar ataxic. My colleague said: "Doctor, I never smiled once." It was really pitiable—rather a cause for tears. Only when well charted, often as the result of repeated or divided studies, is the case open to intelligent analysis, unless its showings have been immediately and consistently normal. The critical tracing of the paths involved is indeed neurologic, but only to be learned by these ear tests, and therefore neuro-otologic.

The great effort of our work has been to simplify and correlate and interpret: not by trying to state vertigo or past-pointing in relation to nystagmus, since nystagmus or even conjugate deviation of the eyes may be lacking or perverted; but to recognize and state that the stimulus is in absolute relation to the direction of endolymph flow. But this cannot always be determined with the "fistula test," hence its limited usefulness. It can be falsely simulated by galvanism, which can act on the nerve trunk or on the nuclei. Hence, our limited use of these tests and distrust of their showings. Enough that by reasonable use of the caloric and turning tests we can learn, often with extreme precision, facts otherwise unobtainable, especially as to subtentorial conditions, of utmost value to the otologist, the neurologist and the surgeon.

Dr. Charles A. Elsberg said he had heard so much that was interesting and instructive that he did not feel he could add much, if anything, to what had been said. He wished, however, to express his thanks to the gentlemen who had come this evening from Philadelphia and presented the results of their large experience.

He had been asked to say a few words on the subject from the surgical standpoint. Of course, from the standpoint of the practical surgeon, it is important to know what value these tests really have. Can they make the diagnosis for us? Can they make a differential diagnosis? When do they fail? These are the questions which the surgeon must have answered—and they have arisen in a number of instances in which he had had these tests made for him.

Of course, before speaking of conclusions, it must be remembered that this work is still in its infancy, and that much more can be learned and will be learned in the future. Therefore, one should hesitate very much before saying anything derogatory to the tests or the results obtained by them.

The questions that have arisen in his mind in the course of the last few years regarding these tests are these:

1. Can we make the diagnosis by their means? Naturally, his remarks would have reference to the diagnosis of those conditions of interest to the general surgeon. His experience has been that, in the first place, they have been very valuable in differentiating between frontal and cerebellar tumors. Thus, for example, the differentiation between a right frontal tumor and a left cerebellar tumor is often difficult. Not infrequently, it has occurred to the most experienced surgeons that a case turned out to be one of cerebellar disease after an operation had been done in the frontal region. In two instances in the speaker's experience, the differentiation could be made by these tests.

In the second place, he had seen a number of children suffering from a nystagmus the cause of which could not be discovered by various specialists—neurologists and others. Finally, cerebellar disease was suspected. In several of these children, the otologist solved the question by showing that it was a congenital nystagmus which had no special significance.

2. What value have the tests for differential diagnosis? He had already spoken of its value in differentiating between frontal and cerebellar disease. He has not, however, obtained much information on the Bárány tests in cases where the diagnosis lay between internal hydrocephalus and cerebral disease with secondary internal hydrocephalus. In hydroceph-

alus the symptoms are often very like those of bilateral or even unilateral cerebellar disease, and in these patients no help was obtained from the tests. In one case the hydrocephalus had been much improved by puncture of the corpus callosum, but even after this the otologist was unable to state from his examination whether there was a primary infection of the cerebellum. It would be interesting to note from Dr. Jones and Dr. Fisher if they have been able to differentiate between cerebellar disease and internal hydrocephalus.

3. He has seen the test fail in disease of the posterior fossa where the symptoms pointed to the opposite side of the cerebellar lobe. As is well known, the tumor in one lobe of the cerebellum may press the other lobe against the petrous portion of the temporal bone and give cranial nerve and cerebellar symptoms on this latter side. In patients of this kind the Bárány test had shown an affection of this latter cerebellar lobe when the real lesion was on the other side. It may have been that the tests were incomplete or not thoroughly done. Perhaps in the hands of Dr. Jones and Dr. Fisher results would have been different. It would be valuable to note whether the failures were mistakes, or whether they were due to some inherent weakness of the tests.

All the foregoing remarks, however, are made in no sense of criticisms of the valuable tests which Bárány had described, which have much furthered our knowledge of vertigo and of the static function of the vestibular nerves.

Dr. Arnold Knapp: It is quite true that nystagmus is generally regarded by ophthalmologists as an associated symptom and of little diagnostic significance. To those, however, who have been fortunate enough to hear Dr. Jones' admirable demonstration and to see his remarkable moving pictures, nystagmus will have assumed a new meaning.

As for the practical value of the vestibular tests in nystagmus and in strabismus, though we have made at the Eye Hospital a routine examination of suitable cases, our experience is far from complete. In the form of nystagmus which is most familiar to us, the socalled amblyopic nystagmus which occurs in children with defective sight, albinism or retinitis pigmentosa, the vestibular reaction has been found unchanged. There is another group of cases of nystagmus in children without any ocular signs of amblyopia who are suffering from congenital lues. In these cases Heine has found the spinal fluid to be under increased pressure. A case which recently came under the speaker's observation was that of a child of four years, who had suffered from right hemiplegia, then convulsions and nystagmus. After lumbar puncture the nystagmus and convulsions ceased.

In the cases of strabismus, those with the ordinary concomitant convergent strabismus, we have not been able to find any change in the vestibular reaction. This does not agree with Bartels, who found a disturbed reaction in nearly half of thirty patients. In two cases of nystagmus combined with convergent strabismus which we recently had the opportunity of examining at the Eye Hospital, the reaction to the vestibular tests was unaffected. Paralysis of the ocular muscles would seem to offer a field for this form of examination, and investigations along these lines have been made by the men in Vienna.

The vestibular tests offer a means of distinguishing between a supranuclear and a nuclear lesion, and if the reaction is normal the lesion must be supranuclear. Supranuclear lesions, however, are usually of associated movements, such as conjugate paralysis, conditions which are generally found in severe nervous disease and do not often visit the eye dispensaries.

This subject is surely a very interesting one, though to my mind it presents many difficulties; still, after listening to Dr. Jones' exposition, we should feel encouraged to persevere in our examinations.

Dr. Isidore Friesner said that in listening to these very interesting papers there would seem to be two topics for discussion. First, a consideration of the physiologic activity of the static labyrinth; and, second, the question which Dr. Knapp had just brought up—a consideration of the basis upon which conclusions from functional tests may be drawn.

With regard to the physiology of the static apparatus, he had so often and at such length discussed this question with his friends from Philadelphia, that it would be passed by at the present, except to say that it is not necessary for one to adhere to the cerebral or the cerebellar origin of the past-pointing in using the pointing test. The origin of this phenomenon is not essential, or at all events not vital to our conclusions. Whether we believe past-pointing to be cerebral (the objective expression of the vertigo), intermediated by the cerebellum, or whether we hold that past-pointing is purely cerebellar, we use the pointing test in determining the disturbances of cerebellar function. From the point of view of the otologic surgeon, the mere presence of labyrinthine function is sufficient to warrant adequate conclusions. Of course, that is evidenced by the presence of hearing and the response to any one of the several stimuli which are applied to the static labyrinth. In making such tests we have all, time and again, seen decided differences in the responses from the two sides; or if we have tested the separate parts of the static labyrinth, we have seen decided differences in the responses from them; but because our conclusions were based entirely on the presence of function, these differences were disregarded.

From the neurootologic point of view, our conclusions are based largely on the intensity of the response and the promptness with which the stimulation arouses an impulse. This point of view is different from that of the otologic surgeon. For instance, with normal peripheral conditions, if we apply the same light separately to each eye, and find that the iris in one contracts promptly and fully, while that in the other contracts sluggishly and incompletely, do we not conclude that there is some disturbance of the reflex arc in the latter? Are we not equally justified in our conclusions if we find such differences after applying stimulation of equal intensity to both static labyrinths, even to the separate parts of each static labyrinth? For it is possible to stimulate calorically the horizontal and vertical canals separately.

Indeed, some recent tests of the vertical and horizontal canals separately, in cases of vertigo accompanying toxic conditions, accompanying purulent sinuitis—one case a compositor with lead poisoning—have indicated the possibility that there may be a retrovestibular neuritis analogous to a retrobulbar neuritis, in that certain fibers of the vestibularis are more susceptible than others; and, further, it would seem that the more susceptible fibers are those conveying the impulses from the vertical canals. The nerves have not been tested galvanically in these tests.

The diagnosis and localization of cerebellopontine angle tumors by the functional tests is, of course, comparatively easy. We, too, have observed in cerebellar pontine tumors the transmutation of the nystagmus impulses. Normally, the cold caloric test on the right side causes a nystagmus directed to the left; in several cases of cerebellar pontine tumor we have seen, following caloric stimulation, an oblique nystagmus or a vertical nystagmus upward. It would seem probable that the cross-pointing with cerebellopontine angle tumors, so well shown in Dr. Jones' "movies," is brought about by pressure on one or both lateral cerebellar hemispheres. Much of this work, as Dr. Jones said, is purely experimental.

Dr. Friesner concluded by saying that he would not wish to be misunderstood, particularly with regard to what he had said concerning retrovestibular neuritis. His statements were in no sense conclusions, but merely suggestions.

Dr. Joseph Byrne, after expressing his appreciation of Dr. Jones' wonderful presentation of the subject, said he himself had done a good deal of work upon the labyrinth. Some of Dr. Jones' statements were misleading, especially in regard to the precise mechanisms involved. Thus, in rotation tests, Dr. Jones said that the eyes are always drawn (long component of the nystagmus) in the direction of the movement of the endolymph, but he failed to state that during the actual rotation there is a primary nystagmus which, upon cessation or slowing down of the movement, gives place to the secondary nystagmus. It is this latter which is of opposite direction to that of the primary nystagmus that the observer studies. Dr. Jones said this secondary nystagmus is due to the continuation of the movement of the endolymph. This is true only in a sense, since Dr. Byrne had observed reversal of the nystagmus —e. g., from the primary to secondary form, with mere slowing down of the movement in rotations. It is well known, moreover, that the lumen of the membranous canals is of capillary dimension, and so precludes the possibility of such a thing as gross movement of the endolymph in rotations. What we are dealing with in rotation is not gross movement of the endolymph, but the most delicate alteration of conditions about the hair cells (slight bending), due to faint alterations of hydrostatic tension. A mechanism permitting of gross movement of the endolymph would be quite useless for fine physiologic adaptations.

Dr. Byrne said that another thing to which he has always taken exception in Bárány's work is the assumption that, in the caloric tests, heat and cold produce their effects by causing movement of the endolymph within the membranous canals. Such movement, assuming that it does occur, implies displacement of the endolymph in opposite directions—e. g., currents of opposite direction arise, which, owing to the minute size of the canals, would, upon continuation of the irrigation, soon cease, owing to the equalization of temperature in the endolymph the whole way across the particular canal involved. This does not occur, as can be readily demonstrated; for the nystagmus persists in spite of prolonged irrigations. Numerous other objections might be urged against Bárány's view on this point, but Dr. Byrne contented himself with one experiment of his which seemed conclusive. It is well known that the labyrinthine receptors, especially those in the semicircular canals, are exceedingly sensitive to all kinds of stimuli—mechanical, electrical, thermic, and even photic. It is also well known that in any posture involving equilibration, tonus impulses are constantly streaming inward from the labyrinthine receptors, and that equilibration itself implies nice adjustments between the impulses coming in from the various labyrinthine receptors on either side of the body. In a word, in the labyrinths we have nicely adjusted balanced mechanisms. Assuming, as Dr. Byrne has done, that within certain limitations, heat exalts and cold depresses nerve function, as is well known to all physiologists, then prolonged cold irrigation should deaden the receptors affected and render them insusceptible to the effects of other forms of stimulation. This was put to the test. After irrigation of the left ear at 68° F. for four minutes the subject was immediately rotated from left to right about the vertical axis. On sudden interruption of the rotation the usual after-nystagmus was entirely missing, whilst the primary nystagmus to the left and against the watch persisted. This experiment, which seems conclusive, was put on record by Dr. Byrne as far back as 1910, but the work has been ignored or neglected.

There were statements in reference to vertigo made by Dr.

Fisher to which Dr. Byrne took exception. Vertigo is not a sensation in the ordinary sense, having a special receptor organ in the labyrinth, as Dr. Fisher states. This conception of vertigo was objected to by Ferrier over forty years ago. Orientation is an awareness or consciousness of our relations in space, and disorientation, of which vertigo is a form, is a disturbance in the sensory elements, etc., upon which this awareness is based. With this disturbance there goes, of course, a certain sense of insecurity, disordered equilibration, etc., which, from the subject's point of view, are the main features of disorientation. But all these do not make vertigo a sensation.

Dr. Byrne wished to call attention to the fact that the labyrinth has other and very important functions in the economy. He had long ago demonstrated its influence upon vasomotor function. Leonard Hill has shown that if a hutch rabbit be held upright it will, in from ten to twenty minutes, go into convulsions from cerebral anemia and die. Cats, dogs and wild rabbits, with their more efficient abdominal musculature, withstand the erect posture for much longer periods. Hill also found that if hutch rabbits be held submerged in water they will withstand the erect posture almost indefinitely without disastrous consequences. These facts are of intense interest where there is question of the erect posture and the influence of the labyrinth on tonus, whether of striped or unstriped muscle.

The labyrinth is also related to the vomiting mechanism. No vomiting center has as yet been demonstrated distinct from the respiratory center or that portion of it in direct relations with the effector mechanisms of expiration. It is probable that the otolithic apparatus (statoliths of Verworn) is in direct relation with the vomiting center. Every physician is familiar with the movements of the nares, lower jar, trachea and accessory muscles of respiration in impending death. These phenomena were matters of observation from time immemorial. Dr. Byrne said he had pointed out that they were all due to the progressively increasing demands made upon the great respiratory pump as an auxiliary to the heart and vasomotor system when the circulation begins to fail in the medullary centers. These phenomena are comparable to the dyspnea of asphyxiation and the general convulsions (both due to increased hydrogen ion content of the blood) that close the scene.

Dr. Byrne pointed to the frequency with which vomiting or gastric regurgitation occurs at or near the point of death. The forced respiratory action and the vomiting are defensive mechanisms. They are the last reserves called out to stave off final dissolution. Disease, of recent years, has been defined in terms of reactions of the organism. In seasickness, which Dr. Byrne has studied extensively, it is probable that the vomiting and vertigo are protective reactions. In rotations and in seasickness Dr. Byrne has shown that one of the first effects upon the gastric mechanisms is closure of the pylorus. This is the first step in a train of phenomena which culminates in vomiting. This latter is a most powerful means of increasing the cardiac output, constricting the arterioles, and enhancing blood flow in general. In seasickness the medullary circulation is restored directly by the vomiting and indirectly by the vertigo which enforces recumbency.

Dr. Jones, in closing the discussion, said he thought Dr. Elsberg would find the following of help in the differential diagnosis between internal hydrocephalus and a lesion of the cerebellar substance: An internal hydrocephalus would as a rule show no nystagmus or past-pointing on stimulating the vertical canals of both ears, but stimulation of the horizontal canals of each ear would produce a normal nystagmus and a normal past-pointing. In a cerebellar lesion, however, no matter which canals are stimulated, we would expect a consistent absence of past-pointing of a certain extremity in a certain direction—for example, an absence of past-pointing of the right arm to the right. Further, if the cerebellar lesion did not involve the brain stem, we would expect a normal nystagmus on stimulation of all the canals.

Replying to Dr. Byrne, the following experiment would seem to prove conclusively that a movement of the endolymph must be accepted as the cause of stimuli from the internal ear: With the head back, douche the right ear with cold water; there occurs a horizontal nystagmus to the left, vertigo to the left, and past-pointing to the right. Without any further douching, incline the head forward; there immediately occurs a horizontal nystagmus to the right, vertigo to the right, and past-pointing to the left. Simply by altering the position of the head we have produced diametrically opposite phenomena. This clinical fact can only be explained by an endolymph movement, first in one direction and then in the other.

Dr. Fisher, replying to Dr. Elsberg's inquiry, said that these tests are valuable insofar as one is confident of his technic and is certain of the anatomic pathways which convey the results of stimulation. It is believed that when the normal ear is stimulated it will produce normal responses. When stimulation fails to produce normal reactions, it is always due to an interruption in these pathways. Such an interruption may be located in the ear itself, or in the pathways, or in the brain centers, and the ability to visualize these pathways, combined with a careful and painstaking technic, enables one to make a correct diagnosis.

As to Dr. Byrne's remarks about vertigo not being a conscious sensation, if you put an individual in a chair with the eyes closed and turn him to the right, he will say: "I am going to the right." Then, after you have turned him ten or fifteen times, and the movement of the lymph has caught up with that of the body, he will say: "I am standing still." As a matter of fact, he is being turned very rapidly, but because his lymph is moving as fast as his body, he is under the impression that he is not moving at all. Then suddenly stop the chair, and he will say: "I am going to the left." How is he to know all this unless it be a cerebral act?

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of February 9, 1917.

Remarkable Development of Mastoid Cells Following Fracture in Childhood.

DR. J. D. KERNAN: The patient was a young man, eighteen years of age. His family history had no bearing on his condition. He had had the usual diseases of childhood, and frequently suffered from colds in the head. Seven or eight years ago he fell four stories, but does not know where he was hit. He could not walk for three months, but does not think he was paralyzed or had any bones broken. Has been hit on the head a number of times. He has always been perfectly well, and has had no earache or trouble of any kind.

Two months ago, by chance, he noticed a swelling behind the left ear, which grew steadily larger, especially when he blew his nose. Before that he had always heard well; now, at times he does not hear so well, and then again hears perfectly. When he presses on the lump he does not hear so well in that ear. When he moves or walks he hears a crackling over the lump, somewhat like crackling in ears when swallowing. He thinks he was feverish once in the last two months. His appetite is good, bowels regular, and general health good.

X-ray pictures were presented for examination.

The examination of the ears gave rather negative results. The hearing of the upper and lower tone notes seemed to be normal. When the enlargement was pressed on there seemed to be a slight diminution in the hearing—a diminution of the air conduction and a lengthening of the bone conduction, but the changes were very slight.

Dr. Kernan said that three possibilities were to be considered as the cause. One, that the enlargement was merely a natural increase of the process of growth in the mastoid cells, such as is seen in the lower animals and especially in the anthropoid apes, where the mastoid cells extend into the surrounding bones; second, the possibility that when he was injured the bone was broken in such a way as to give freer access than usual to the mastoid from the middle ear, and the blowing of his nose has caused pressure of the air into the mastoid and continued the process of cell formation; and third, that there may be some new growth, such as angioma or sarcoma, growing from the region of the middle ear and gradually boring its way through the bone.

DISCUSSION.

Dr. Law said that he had brought the plate down to find out what the members of the section thought about it. He himself did not know what to say about it. The picture on the plate certainly seems to be that of an enormous development of the mastoid cells. He did not think that it was a new growth, primarily, as it did not seem that a new growth would have eroded the bone with such a clear cut outline of cells; he did not know of any new growth that would do that. The pneumatic area, if one might so call it, seems to be in direct connection with the mastoid on that side, and extends to within half an inch of the opposite mastoid. One could conceive of a new growth superimposed on the mastoid, as accounting perhaps for the lump on the back of the head, but so far as he could make out from looking at many mastoids, this seems to be a mastoid structure. If it is anything else, he hoped someone would tell what it is.

Dr. Lewald thought the accumulation of air beneath the scalp the most difficult to explain. It might be called a pneumocephalocele, but that was only a name; and he had not the faintest idea what to say concerning the origin of the condition.

Dr. Lederman said he had never seen anything like it, and would like to know what Dr. Kernan proposed to do in the line of treatment. In his opinion, Dr. Kernan's explanation of the injury as being the cause of the condition seemed the most probable, from the fact that the young man can blow up and enlarge the mass, showing a direct communication from the middle ear. As he seems to be quite comfortable, it would seem questionable whether surgical interference would be justified, as it might do harm.

Dr. Hays said that the case reminded him of a bone sarcoma, which is frequently seen in young children. In these cases there is a peculiar egg shell crackling which is distinctive and which can be felt in this case. From the X-ray plates and from the history of the case, it seemed to him that there was no definite anatomic connection with the mastoid bone. The X-ray showed the cell cavity extending over almost all the bones of the cranium. Of course, it is possible that these cells connect with the mastoid cells and the middle ear itself, so that in forcing air into the ear such air could be sent through these various cells.

Dr. Hays said that he had seen cases of sarcoma of the antrum in young children, in which bone would become very thin and would pit and crackle on the slightest pressure. Such sarcomata are not very malignant and do not form metastases very quickly. He said he felt that this was a case of pathologic change in the bones which connect with the mastoid cell.

Dr. Herzig asked whether the X-ray was taken of the left side, or of the right side, or both. To which Dr. Law replied that pictures were taken of both sides, and the mastoid cells were within one-half inch of the other side.

Dr. Haskin asked if anyone had any idea of what caused the crepitation.

Dr. Wilson, replying to Dr. Haskin, asked if it might not be the air in the scalp.

Dr. Haskin said that it felt as though there was a thin plate of bone that could be pressed down.

Dr. Wilson said that the crepitation disappeared when the air disappeared, and there was the distinct feeling of air under the scalp after blowing the nose.

Dr. Law said that with the examination made with the stereoscope there was no thin area as compared with the rest of the mastoid. In other words, the floor of this soft mass throws as dense a shadow as the rest of the area, so that if there is an increased thinness of the bone, it does not show in the plate.

Dr. Kernan said that as he understood the egg shell crackle in bone sarcoma, it is due to a thin plate of bone over the sarcoma, but he could not make out any bone over this soft tumor; it seems to lie directly under the skin without any overlying bone at all. What few cases of sarcoma of the bone he had seen always showed the bone about the sarcoma in a state of rarefaction—thin, diseased looking bone. The bone in this case is extremely healthy looking except for the presence of the cells. He did not feel that he could accept the diagnosis of sarcoma, on account of the healthy appearance of the bone in the picture. As to the crackling, one sometimes sees the same thing in knee joints where there is inflammation. The synovial membrane being thickened will crepitate in the same way as this swelling when it is palpated; and there might be some inflammation present in this case, with thickening of the walls; or there might be, as the result of the old injury, a few loose bits of cartilaginous material which crepitate like a loose cartilage in the knee joint. He was inclined to consider the condition to be a result of the injury, which opened the way for undue extension of the mastoid cells.

A Case of Unilateral Aural Microtia With Unusual Features.

DR. WM. H. HASKIN: The patient was a boy of eleven years, who had recently come to the clinic. Dr. Haskin said that the condition spoke for itself, and there was little to be said about it excepting that it was a case of congenital microtia with a very peculiar lack of development of the inferior maxilla. There was no zygomatic process, the bone ending abruptly at the malar process. There was some hearing on the defective side. With the Bárány apparatus in the good ear, the patient recognized the difference between a whistle and the loud ringing of the fork, and he could hear loud shouting; he recognized that it was a voice, though he could not detect words. Several X-ray pictures had been taken, and could be seen by any who were interested in looking at them. Rotation in the chair showed an apparent loss of function of the static labyrinth. Rotated to the left, there was a long nystagmus to the right, and vice versa.

Dr. Haskin said in all the years that he had been on the service he had not seen any cases of microtia until recently, when this case and another one had appeared. The other patient was a man with a deformed ear in the normal position, and his rotating test showed the same thing—when rotated to the affected side, he had a long nystagmus to the right; but when rotated to the right, there was nystagmus to the left of only five seconds.

He realized that nothing could be done for this boy. There was no appearance of ossicles; from the stereoscopic plates there seemed to be a lack of development of the greater wing of the sphenoid. If there was any wing forming the floor of the middle fossa, it was very, very thin.

Dr. Page said he did not think it was an operative case, as no case with one good ear is, and he did not think anything could be done for it. It was an extremely interesting condition. He did not understand why there should be the associated maldevelopment of the jaw. It is known that in cases of microtia there is usually a lack of development of the anterior wall of the canal, and of the anterior superior wall of the tympanum, with drum membrane and ossicles possibly lacking, but with the footplate of the stapes present, the internal wall of the tympanum present, and a practically good labyrinth present. Alexander gives as the reason for frequent failure in operations for microtia the attempt to enter along the line of what seems from palpation to be the line of the external auditory canal, as in this direction one encounters the jaw or the facial nerve. That is why Jaeger, in 1830, devised the operation of entering the mastoid antrum and working forward and letting the sound wave reach the stapes through the aditus. A point of diagnostic value made by Alexander is that where there is lack of development of the internal ear, there is almost always associated with it some facial paralysis. From the appearance of this case, he was inclined to believe that there was neither tympanum nor ossicles, but a stapes and internal ear.

Dr. Lederman said that a number of years ago he saw a bilateral case of this kind—i. e., a child with a similar deformity on both sides. This child could hear a loud voice and other loud noises The father and mother were both healthy and robust. When looking up the literature at that time, he had found that most men who had given attention to the subject agreed that operation offers no hope in these cases, as there usually existed a malformation of the middle ear. The late Dr. Hermann Knapp had spoken to that effect before the section a number of years ago.

Dr. Norton L. Wilson said that he had had a somewhat similar case, but there was not so much deformity of the jaw. The child had ossicles, but the closure was complete to the middle ear. The child had a little hearing, and he had endeavored to help the condition, but without any result. He was neither able to improve the hearing nor to keep the newly formed canal open.

Dr. Kernan said that the case was of great interest from the embryologic point of view, since the ossicles develop as part of the lower jaw from Meckel's cartilage. Therefore, in such a case as this where the caudal end of the lower jaw is undeveloped, one would expect that the incus and malleus would also be undeveloped. The stapes is developed from the proximal end of the hyoid arch, so that one would expect in this case to have the stapes present, and some hearing, whereas the conducting apparatus formed from Meckel's cartilage would be absent. The inner ear develops independently, by itself, and that might be perfect, while the middle and external ear are entirely absent.

Dr. Page said that he was just about to ask what was the relation of the development of the jaw to the development of the tympanum and ossicles. Politzer states that (according to Gradenigo) the footplate develops in the wall of the labyrinthine capsule.

Dr. Kernan said there had been a great deal of discussion on this point, but he was under the impression that it had been decided that the whole bone develops from the proximal end of the hyoid arch—that it is one structure; the footplate does not develop separately from the rest of the stapes.

Dr. Haskin said that an especially interesting point about this case was that the boy had no zygoma. The lack of development of the lower jaw probably resulted from the fact that, as there was no glenoid fossa, the child had never been able to use that side of the jaw for mastication, and there had been no stimulation to produce the growth. He further said that he had looked up many histories of microtia cases, and had not found any described as in any way resembling this one. The position of the cartilage, well down on the cheek, rather than in the normal position, is the chief peculiarity. He had made pressure over where the internal ear should be, to see if there was any vertigo, but had not succeeded in getting any response. There were a great many interesting points brought out by a study of the plates.

Paper: Clinical Observations of a New Iodin Preparation (Iodosan)

By M. D. Lederman, M. D.

DISCUSSION.

Dr. Heller said that all had at some time felt the need of having a good powder to blow into the ear for the treatment of chronic discharge, but had been limited to boric acid, which was nontoxic but had the unfortunate quality of not dissolving very rapidly, and consequently caking in the canal. The other antiseptics, bichlorid of mercury, iodin, and nitrate of silver, do not lend themselves to being used as powder; argyrol has been used frequently, but it is a nasty preparation, has little therapeutic value, and makes everything look black and as disgusting as possible. He had used this iodin powder, described by Dr. Lederman, on five or six cases, and felt that it was a distinct help in the treatment. It is readily absorbed. In one instance in which he used it in full strength there was a fair sized perforation, and the patient had a little pain, and nothing was done to counteract it; the man returned in an hour and said that he could not stand the pain, and on looking at the ear there was none of the powder left—it had all been absorbed excepting a little on the skin and outside of the meatus. Dr. Heller said that he was absolutely sure that it was absorbed, and was also sure that it was iodin, for he had had a little of the powder at home, and failing to realize that it ought to be kept in a glass stoppered bottle, found that the cork had been disintegrated at the lower end and had fallen out. He then showed a bottle where the cork had been used for only a few hours and that it was distinctly injured and discolored by the iodin. He repeated his belief that the powder was a valuable addition to the therapeutics of the ear.

Dr. Otto Glogau said that he had been using this preparation in chronic suppurations of the middle ear and maxillary antrum for over two years. Some of the patients complained of pain when a ten per cent mixture was used, so he employed instead a three per cent mixture, and had had no trouble since It was necessary in using it to remove first all diseased tissue such as granulations, polypi, necrosed ossicles, etc. In chronic suppuration of the maxillary antrum he had used it after operation and found it very valuable. It becomes absorbed very rapidly and does not cake up the wound. Combined with surgical treatment in these chronic middle ear suppurations, this powder will be a very valuable aid in our therapeutics.

Dr. Carter asked whether the preparation was a mechanical mixture or a chemical compound. He also asked Dr. Lederman how he accounted for the fact that while there is only five per cent of iodin in the preparation, the remaining boracic acid powder does not cake as when plain boracic acid is used.

Dr. Lederman said that he was not enough of a chemist to describe the exact technic in the manufacture of the preparation, but that it was probably more of a mechanical than a chemical combination, as the iodin is very loosely bound with the boric acid and separates from it very readily, which is of especial advantage. In other preparations the iodin is so tightly bound that it is not readily liberated. This condition must be borne in mind in applying the powder, and one should be sure that the powder is finely divided before being blown into any cavity.

In a case still under treatment, the patient was a young man who had had a chronic suppuration for a number of years. In one ear, two-thirds of the drum was gone with granulation tissue present. The powder seemed to have some action on the granulation tissue in the middle ear, and when a cotton covered applicator was passed over the granulation tissue it was found to be friable and was easily removed. He has been under treatment for six weeks, the discharge in one ear has ceased and the drum has healed over. In this ear the perforation was at the posterior margin and involved the posterior third of the membrana. The other ear is almost dry.

The advantage claimed for the powder is that the iodin content is easily given off, and there is no danger of retarding the drainage. He has used it in a number of mastoid wounds, and found that it hastened the healing. It had also been used by a surgeon in vaginal gonorrhea, and in two instances it had been used on a suppurating breast, and in a few applications the fistula which the surgeon had been trying to combat were healed. It has been used abroad in cases of suppurating antrums with very good results. The preparation is not yet on the market, but can be obtained in small quantities at Nauheim pharmacy. Five per cent has been found to be sufficiently strong to be effective, and in fact he generally uses a two per cent mixture, which seems to do the work very satisfactorily.

Dr. Law asked if an X-ray plate had been made after the condition cleared up, in the case mentioned.

Dr. Lederman said that the patient lived out of town and had not been seen for some time, and no plate was made after the discharge ceased.

Dr. Haskin suggested the employment of a very strong salt solution in cases of chronic suppurative ears, letting it remain in the ear. He had done this in several cases, following the suggestion of Sir Almroth Wright in treating suppurative wounds, and the result was very remarkable. By filling the ear up with a strong solution of salt and letting it remain in for half an hour, old cases of foul smelling ears would clear up very surprisingly. If the solution disappears through the eustachian tube, fill it up again.

Paper: Report of Thirty-seven Cases of Perisinus and Extradural Abscess—Lantern Demonstration.

By H. B. Blackwell, M. D.

DISCUSSION.

Dr. Hays said that the speaker had not presented anything especially new on the subject of perisinus or extradural abscess, but only more about an operation which he has been performing for a good many years. No one questions the fact that a majority of the perisinus and extradural abscess cases are what might be termed latent, having existed for a considerable length of time, varying from four to six weeks or more —all of those in which there is a diminution of the acute symptoms and the diagnosis is made on the general looks of the patient, often corroborated by the X-ray picture. All agree that the most serious cases are those that have extended over a considerable length of time and show destruction of the bone, the sinuses or the dura. A majority of cases get well, and Dr. Hays said that in discussing the subject at a former time, he had said it would seem that although the condition had continued until there was considerable destruction of bone, the bacteria apparently became attenuated in virulence. Often these cases heal more quickly than the acute cases, which do not heal up in two or three weeks.

Dr. Hays said that Dr. Blackwell was exceedingly fortunate in having such excellent results, losing only one patient out of thirty-five cases. In regard to the question of operation, Dr. Hays said that six or seven years ago he had described an operation which he had termed primary suture of the mastoid wound. At the time he was very severely condemned for doing it. It consisted mainly of closing the wound and putting in a wick of gauze, but the results have justified the procedure in certain cases, and the cosmetic results have been excellent. Anyone who has Dr. Blackwell's dexterity can go down into the attic and expose the malleus and the incus, but that is unnecessary in the majority of cases—and the best evidence of that is found in the cases he had reported, in which he had never seen the head of the malleus. In acute cases of mastoiditis, we should let the aditus and the attic alone as much as possible. The main destruction of bone is in the mastoid cavity, and if that is cleaned out thoroughly and drained by a large wick of gauze, the case will get well nicely.

Dr. Page complimented Dr. Blackwell on his excellent results, and said in regard to closing the wounds, however, he had had an experience similar to that reported by Dr. Hays. This occurred with a simple mastoid operation performed for mastoiditis with a perisinus abscess in a young man. His wound was closed down to a small drain at the lower end. The drain was removed in three or four days, and there was union and an apparently clean cavity at the end of a week. He was allowed to get out of bed on the fifth day and allowed to leave the hospital on the seventh day, not having had any rise in temperature. He was directed to report to the hospital in two days, but to return the next day if he felt ill. The next day he felt ill, but stayed at home, and on the day following came to the hospital with a temperature of 102°. He was readmitted to the hospital for exploration of the wound, which, to external appearance, was free from inflammation. The skin flaps which had been reflected and replaced at the time of operation were found adherent to the sinus wall, and localized areas of infection were walled off by adhesions, with no communication with the lower angle of the wound. An infected clot was in the sinus, and it was found necessary to resect his jugular. Recovery occurred only after a stormy career with a femoral phlebitis and other complications. Dr. Page said this had cured him of taking any chances simply to get a rapid recovery.

While he closes the wound down to the lower angle, he protects the whole exposure of the dura and sinus to the farthest angle of the wound with gauze strips saturated in salt solution, with their ends at the lower angle of the wound, in order that no areas of infection can become walled off by adhesions between the integument and the knee of the sinus. More recently he has been using Dakin's solution in the place of normal saline, and the gauze is kept in place not longer than three days, and it may be removed the day after the operation if conditions appear unfavorable. For the next five or six days the wound is dressed and the extremities of its cavity are examined through a large speculum, and if everything is clean and there is no discharge from the canal, the skin is allowed to grow together at the end of a week or two. At the first indication of infection in the wound, however, it is opened sufficiently to properly avoid danger to the patient.

Dr. Page said, however, that he had never had such a percentage of success as that reported by Dr. Blackwell, in which all thirty-five cases (if he understood him correctly) healed within from fourteen days to three weeks. This was to him a very remarkable record.

Dr. Lederman said that some seventeen or eighteen years ago he had heard Dr. McEwen discuss the treatment of brain cases at the otological meeting in London, and had been deeply impressed by his remarks on the handling of the dura. McEwen stated that he did not remove the granulations on the dura, as they were a protection against further infection. Ever since then, in operating a perisinus or extradural abscess, he has carefully avoided injuring the barrier which nature has set up, which is frequently destroyed in the attempt to make a clean wound. In operating upon acute mastoiditis cases, he is still satisfied to clean out the antrum and adjacent cells which are involved, but does not attempt to disturb the aditus or middle ear. If the antrum and mastoid cells are thoroughly cleaned, the middle ear condition will promptly subside.

DR. HERZIG complimented Dr. Blackwell on the excellent results secured, and asked how much time was allowed to elapse after doing a radical case before the first dressing was made—i. e., closed the wound. He also wished to know if he was correct in understanding Dr. Blackwell to say that the wounds healed in fourteen to twenty-one days, and how often the dressing was replaced.

Dr. Herzig said that he had sewed up three cases, closing the wound completely, with the exception of a small drain. One of these healed in four weeks. That one was found years ago. In the second case, a double mastoid, one side did not heal at all, the other side healed in four weeks. The third case did not heal for three months, but finally closed up. He wished to know how often Dr. Blackwell replaces the dressing, and if he packs it up to the antrum. The closed wounds in all of these cases show a strong covering, and no sinking in to any extent.

DR. HASKIN said that Dr. Blackwell was certainly to be congratulated upon his excellent results. The paper was really devoted to two questions—the perisinus abscess and the method of operation. Dr. Haskin said he was inclined to doubt the wisdom of opening the attic in acute cases. After opening and securing drainage, it would seem much safer, in the great majority of cases, to keep out of the attic, for the attachments of the incus to the aditus are very easily injured, and there does not seem any advantage in stirring up avenues of infection. In the great majority of cases, when the infection is removed from the mastoid process the condition will subside.

Dr. Haskin said that he employs another method of healing wounds, which to some might seem rather radical, but that he never sews up wounds in either actue or radical mastoid operations. He makes as small an incision as possible, puts in only what dressing is absolutely necessary, and keeps the wound open for five or six days, and then cleanses the wound every day for a few days until the bone is covered with granulation tissue, and then leaves off all dressings. It is astonishing how rapidly they heal under this treatment, and how beautifully the edges heal without any depression or deformity. He has never had any bad results from this method of treatment. In the radical mastoid operation he employs the same procedure, taking down the canal wall clear to the ridge, splitting the narrow end of the canal about one-half inch, and packing the ends wide open, leaving the wound open behind, just as in a mastoid wound. After the bony wound has become thoroughly granulated, all the dressing is left off, and you are perfectly sure that the flap ends at the inner ends of the canal are adhered. When you look into the ear afterward, it is normal, so far as the canal is concerned. Dr. Haskin said it was his experience that patients so operated upon have better hearing and heal more rapidly. In the great majority of cases they are dry in three to four weeks.

Dr. Hays said that when he had presented the question of primary suture of mastoid wounds some years ago, he had made a remark that holds good today. In these cases of simple mastoid, where there is no exposure of dura or sinus, he closes up the entire wound except the lower angle. Only one small piece of packing is inserted to reach from the antrum along the posterior wall to the lower edge of the incision. This packing is removed at the end of thirty-six hours. No packing is used after this, but each day a probe is passed into the wound up as far as the antrum. The secretions in the mastoid cavity are expressed by massaging the wound from above downward.

Dr. Hays said that if any otologist can convince him that he is any better off for looking at the wound every day, and interfering with what nature is trying to do, he would stop this procedure. It is a very simple matter to open the wound, even on the fifth or sixth day, so that we can inspect the parts underneath. The trouble is we do not allow nature to do enough for us.

Dr. Hays said that many of his cases in which he had performed the simple mastoid operation had completely healed up on the ninth or tenth day, so that a small strip of adhesive plaster was all that was necessary over the wound.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of March 9, 1917.

Demonstration of Anatomic Specimens of the Temporal Bone.

Dr. Wm. M. Dunning expressed his gratification at the opportunity afforded him of exhibiting "his bones" to the members of the section, though he hardly felt that he could add anything of interest to their knowledge of the anatomy of the temporal bone. His part of the program was merely an exhibit of prepared specimens of the temporal bone, but the collection was somewhat unique in that it covered all the stages of growth from infancy to adult life, showing the appearance of the facial nerve on the surface at different ages. At birth the facial nerve emerges posterior to the drumhead and extends downward in a shallow groove as the mastoid develops, and usually occupies its normal adult position at the age of six.

The collection consisted of about sixty bones, which had been assembled during a period of some fifteen years. Dr. Dunning directed especial attention to the dissections of the semicircular canals and the various and unusual developments of the mastoid cells, some of them extending far forward and others into the occipital condyle. As a rule, at birth there are no mastoid cells except the antrum, which is sometimes as large as in the adult. The cells are probably developed between the first and sixth years, although Gray claims that they continue to develop until puberty. It is also unusual to find small cells at the mastoid tip, as stated by Gray. Large cells are much more usual, and not "cancellous bone containing marrow."

The collection was on exhibit, and the bones would speak for themselves. The first group—including sixteen bones—showed the development of the temporal bone from birth to adult life, and the position and size of the facial nerve at advancing ages. The next group represented different operations, and included some twenty specimens. The remaining specimens illustrated different anatomic conditions of the mastoid, and included a number of interesting instances of fracture.

Presentation of Specimens of Temporal Bone, Excavated to Show the Relations of Important Structures Encountered in Performing the Mastoid and Labyrinth Operations.

Presented by Dr. W. A. Scruton for Dr. A. B. Duel. The main points which Dr. Duel wished to bring out in presenting these specimens were the following:

The relation of the styloid foramen to the mastoid tip in infancy, childhood, and adult life. The series of bones, mounted in sequence from the newborn to adolescence, show with great emphasis the real danger of injury to the facial nerve which confronts the operator in elevating the periosteum in the region of the tip in young children.

A specimen is shown of the temporal bone in the newborn. In this it may be noted that the petrous and squamous portions are separate, also the ring. By reason of this multiple genesis, dehiscences are known to exist even in bones which have reached the period of total ossification; and under such circumstances it is quite conceivable that the simple operation of opening the membrana tympani (myringotomy) may deal a death thrust in the dura or the jugular bulb. Dr. John R. Page has reported a case of injury to the jugular bulb in myringotomy before this section.

The specimen consisting of the entire half of the head with the contents of the temporal bone worked out in detail is recommended for teaching purposes. The student may place this upon a table and by placing himself in position for operating, a study may be made of the planes and angles of approach to the landmarks in the position in which they will be found in actual practice. Study of small specimens only, viewed from all aspects, is likely to result in confusion of the student's orientation when he looks for the landmarks in the sequence of operative procedure.

The specimen consisting of the entire head containing a wax model of the brain is invaluable for teaching purposes, because the temporal bones, which are worked out in detail, are removable, permitting a view of the areas of the brain corresponding to any portion of the mastoid field.

Other specimens presented show the internal ear cut out in various fashions to show some particular detail.

DISCUSSION.

Dr. Haskin said that one of the points brought out in the specimens presented was the position of the facial nerve in regard to the tip. It comes out almost on a level with the superior wall of the canal during infancy, and it is very common for the facial nerve to be injured at that point in operations upon children. In studying mastoiditis he had always been especially impressed with two or three points, one of them being the fact of the depth of the cells between the sinus and the posterior canal wall—the depth to which they extend down into the mastoid. In some large mastoids these cells continue on, under or above the jugular bulb, and may extend clear through into the condyloid process or the occiptal bone. In his collection were cells where a pencil could be passed through to a condyle. Most operators in doing a mastoid take off the tip cortex, but do not take off much of the floor of the tip, yet examination of the bones shows that the cells extend very deeply beyond that point, and in severe cases these parts are not cleared out deep enough, for fear of injuring the facial nerve.

Another interesting point is the relationship of the jugular bulb with the middle ear and with the labyrinth. In the majority of cases one finds the jugular bulb is in contact with the floor of the labyrinth itself. There is very little if any space between the labyrinth and the wall of the jugular bulb. It is remarkable to realize the great number of cases in which this is so.

Another important point is the position of the facial nerve. Dr. Berens possesses the only specimen that Dr. Haskin has ever seen that has the facial nerve going obliquely across the tip of the mastoid; in operating upon this case the nerve was severed. Dr. Duel has several specimens showing dehiscences in the facial canal of the middle ear. That is not uncommon. Quite a large proportion of cases have dehiscences in the facial canal just at the ridge. To a man who has not done much surgery in the mastoid, there is more danger in the descending portion of the facial, for the canal is exposed, and if one gets too close to the canal wall in following these numerous and deep cells, he is liable to injure the nerve in the descending portion; everyone looks out for the aditus and avoids it.

In children the facial nerve at first comes out near the superior wall of the annulus, and one watches for the development of the mastoid tip, the facial canal becomes deeper, or rather the mastoid develops over it and protects it.

Another point is the close contact of the jugular bulb with the floor of the tympanum in many instances. The roof of the bulb is really the floor of the middle ear.

Still another important point is the question of the carotid canal, which forms the floor and part of the anterior wall in the tympanum and the middle ear. By holding the specimens up to the light, one can see the dehiscences in the floor of the carotid canal, which show how easily one could puncture the artery in these positions.

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XXXVI.

A WAX MODEL OF THE NASAL CAVITY AND PARANASAL SINUSES.

BY

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The morphology and topography of the complicated system of paranasal sinuses are very inadequately shown by the usual anatomic methods of dissection, sections or skeletal preparations. Models of these structures are, therefore, highly desirable. Casts or corrosion preparations have been attempted with varying degrees of success by Rauber ('73), Braune and Clasen ('76), Siebenmann ('91), Brühl ('00), and others. The figure of the cast of the accessory nasal sinuses by Sieur and Jacob (Testut and Jacob, Figure 358) and the models of the sphenoidal sinuses made by Loeb ('12) bring out so vividly the size, shape and relations of these parts, that they suggested the desirability of making a cast forming a model of the entire nasal cavity with the paranasal sinuses. The object of the endeavor was to reveal more clearly the complicated form and relationships of these important structures. The nasal pharynx, on account of its intimate relation to the nasal cavity, was also included in the model.

A well developed head was selected, the entire body having been hardened by intravascular injection of formalin. The subject was an adult male, age and previous history unknown. Life-size drawings were made of the frontal and lateral views of the head. A horizontal line (as an alignment mark) was drawn on the surface at the level of the zygoma, to aid later in assembling the sections. The head was then cut into frontal (coronal) sections, each about one centimeter in thickness. These sections were carefully cleaned and studied with special reference to the form, relations and communications of the various sinuses. By means of outlines traced through glass plates, drawings were made of each section. In these drawings the various cells were identified and their communications noted.

Each section was then placed upon a glass plate and a cast made of that portion of the nasal cavity, sinuses, etc., contained in it by pouring into them melted beeswax. A heated glass pipette was used for this purpose, and care was taken to see that the wax penetrated thoroughly into all the smaller cells. The alignment marks on the surfaces of both sides of each section were connected by lines scratched across the corresponding surfaces of the wax. These lines served later for orientation, when the wax slabs from each section were piled together and fused to form the whole model. Fine brass wires were also embedded in the wax wherever necessary to attach isolated cells and to preserve their proper relations, or to strengthen weak points. The tissue around the wax casts was then carefully removed, and the individual wax sections were superimposed, using the alignment marks previously mentioned. Additional wires were then embedded in the wax to unite the sections, and also wherever it seemed necessary to preserve relations or to strengthen the model. It was found that the wax had penetrated very well, and, though great care was necessary, no special difficulty was experienced in assembling the sections. The junction lines between the wax plates were obliterated by a heated needle, and the surface of the model carefully smoothed.

The model was then mounted and given a coat of white paint. The photographs of the various views were then taken. Finally the model was finished by painting in various colors to represent the different parts. The nasal cavity remained white, the frontal sinuses and nasofrontal ducts were painted blue, the lacrimal sac and nasolacrimal ducts yellow, the maxillary sinuses brown, the anterior ethmoidal cells red, the middle ethmoidal (bullar) cells deep pink, the posterior ethmoidal cells light pink, the sphenoidal sinuses green, and the nasal pharynx grayish white.

The general orientation of the model is shown in Figures 1 and 2, representing the relations seen in the anterior and lateral views of the head as a whole.

The various parts shown in the model will now be considered briefly in order. To understand the relations, it must be kept constantly in mind that the model represents a cast of the cavities, or, in other words, the view which would be obtained if the parts surrounding the mucosa of the nasal cavity and sinuses were dissected away so as to reveal their external form and relations (as in Killian's dissections). The solid parts, such as the nasal septum and conchæ, are, therefore, merely negatively represented by spaces in the model.

I.—THE NASAL CAVITY AS A WHOLE.

The general form and relations of the nasal cavity as a whole are shown in all of the eight figures. The length of the nasal cavity from the nares to the choanæ, measures about eighty millimeters, or ninety-five millimeters to the posterior wall of the nasal pharynx. The maximum height is fifty-four millimeters, measured from the floor to the roof of the meatus nasi communis, or eighty-seven millimeters if measured to the upper part of the frontal sinuses. The maximum width, including the maxillary sinuses, is seventy-three millimeters. Of the nasal fossa proper, the width of the roof (meatus nasi communis) is two to three millimeters, of the floor (maximum, including the inferior meatus), about seventeen millimeters on each side.

The nasal septum (s), corresponding to the space between the two nasal fossæ, is shown in figures 5, 6, 7 and 8. It is asymmetric, with a marked convexity to the right, best shown in the superior view (figure 7).

The vestibules (v) are also asymmetric, the right bending lateralward at a marked angle, as shown in figures 5, 7 and 8. The right naris (N) is also displaced laterally and somewhat enlarged. These abnormalities may be due in part to a postmortem artefact.

Although it does not appear clearly in the figures, the floor of the left nasal fossa, in the region of the maxillary sinus, extends about two millimeters lower than the floor of the right fossa. The maxillary sinus in its lowest part on each side is only about one millimeter below the floor of the corresponding nasal fossa.

II.—THE MEATUSES AND CONCHÆ.

In general the meatuses and conchæ are only partially visible in the models, being concealed by the various sinuses, especially by the maxillary sinus. A cast of the nasal cavity proper (without appendages) is shown in lateral view by Rauber-Kopsch (page 182, figure 229). This figure, showing the meatuses in their entire extent, is interesting for comparison with the present model. Figure 358 in the work of Testut and Jacob is likewise valuable for comparison, showing a lateral view of the accessory nasal sinuses. The colored plates of the dissections by Killian ('03) may also be mentioned in this connection.

The superior meatus is visible to a slight extent in the lateral and posterior views (figures 3, 4, 6, sm), just below the posterior ethmoidal cells, with which it is closely associated.

The middle meatus is visible to a slight extent at its posterior extremity, behind the maxillary sinus (figures 3, 4, 6, mm). Anterior to this the middle meatus proper is almost entirely concealed.

The infundibulum, however, forms a prominent ridge, visible above the middle portion of the maxillary sinus (figures 3, 4, i). Just above and behind the infundibulum is a depression corresponding to the bulla, especially well shown on the right side (figure 4, b). The communication between the infundibulum and the maxillary sinus is also clearly shown on the model (figure 4, c). The relations of the infundibulum to the nasofrontal duct are mentioned later, in connection with the frontal sinus.

The inferior meatus is almost completely covered laterally by the maxillary sinus, excepting small portions at the anterior and posterior extremities of the meatus (figures 3, 4, 5, 6, im).

The nasolacrimal duct in lateral view is concealed for the most part by the anterior portion of the maxillary sinus (figures 3, 4, nd). On the right side it is visible throughout its entire extent in the anterior view (figure 5, nd). It opens inferiorly into the inferior meatus, about twelve millimeters from the anterior end of the meatus (twenty-eight millimeters behind the nares), and under cover of the maxillary sinus. Superiorly the nasolacrimal duct is continuous with the lacrimal sac (figures 3, 4, 5, ls). The total length of the duct and sac is about thirty-two millimeters. The portion above the maxillary sinus, which may be considered as belonging chiefly to the lacrimal sac, measures nineteen millimeters in length on the right side, and seventeen millimeters on the left side.

On the left side, the lacrimal sac and nasolacrimal duct present an interesting variation, the lumen being distended to a diameter of five by nine millimeters (flattened mediolaterally). On the right side the duct is nearly cylindrical in form, with a diameter of about three millimeters. (Even this diameter represents a moderate distention of the lumen.) No cause for the abnormal distention of the left duct and sac could be found in the original preparation. The aperture of communication with the inferior meatus was also dilated. The lumen was empty; the lining mucosa was smooth, and no traces of inflammation were visible. The lacrimal sac on each side is nearly uniform in caliber with the nasolacrimal duct of the corresponding side.

The conchæ are represented in the model by surface depressions between the corresponding meatuses, and, like them, are largely hidden by the maxillary sinuses. The anterior extremities of the inferior concha, and the posterior extremities of the anterior and middle conchæ, are visible in the various views of the model (figures 3, 4, 5, 6, ic, mc). The superior concha is concealed by the posterior ethmoidal cells.

III.—THE MAXILLARY SINUSES.

The maxillary sinuses are prominent and relatively long anteroposteriorly (figures 3 to 8, ms). The measurements taken from the model, together with the averages (and range) obtained by Loeb ('09) on thirty sinuses, and by Schaeffer ('10) on ninety sinuses, are shown in the following table:

	Antero-posterior mm.	Supero-inferior mm.	Medio-lateral mm.
Right sinus on model	42	31	20
Left sinus on model	44	33	20
Schaeffer's average (and range)	34 (15-47)	33 (21-50)	23 (12-40)
Loeb's average (and range)	38 (17-42)	38 (17-47)	23.8 (8-33)

The relations of the maxillary sinuses to the meatuses, conchæ, and nasolacrimal duct, and the communication with the infundibulum (best shown in figures 4 and 7, c) have already been mentioned. As shown in the anterior view (figure 5), the lowest part of the floor of the maxillary sinus on each side is but very slightly (about one millimeter) below the level of the corresponding floor of the nasal fossa. Both sinus and nasal fossa extend about two millimeters lower on the left than on the right side, although the floors of the nasal fossæ reach the same level more posteriorly, toward the choanæ.

IV.—THE FRONTAL SINUSES.

The frontal sinuses (figures 3 to 7, fs) are below the average in size. The measurements taken from the model, together with the averages (and range) observed by Loeb ('09) in thirty sinuses (fifteen heads), are given in the following table:

	Supero-inferior mm.	Medio-lateral mm.	Antero-posterior mm.
Right sinus on model	29	25	10
Left sinus on model	33	24	13
Loeb's average (and range)	34 (14-51)	23 (7-42)	21 (9-33)

As seen in the front and rear views of the model (figures 5 and 6), the septum between the two sinuses is oblique. The right sinus is somewhat smaller and more irregular in outline. The left sinus has a small rounded diverticulum, best shown in the lateral view (figure 3, d). In the superior view (figure 7, fs), the frontal sinuses appear blurred and enlarged, on account of nearness to the photographic lens.

The nasofrontal duct on each side passes nearly vertically downward from the frontal sinus to communicate with the anterosuperior extremity of the infundibulum. The nasofrontal duct on the left side is almost completely hidden by the anterior ethmoidal cells. On the right side it is partly visible (figures 5, 7, n). The developmental studies of Killian ('96), Schaeffer ('10), and Davis ('14), however, have shown that the nasofrontal duct rarely represents a direct continuation of the infundibulum as it is usually described in the textbooks of anatomy.

V.—THE ETHMOIDAL LABYRINTH.

The ethmoidal labyrinth presents on the model a mass of irregular cells, of varying size and shape. They are especially well seen in the lateral and superior views of the model (figures 3, 4 and 7), from which it is evident that the two sides are not symmetrical.

The measurements of the ethmoidal labyrinth on the model, together with the averages (and range) obtained by Loeb ('09) for thirty labyrinths (in fifteen heads) are given in the following table:

	Antero-posterior mm.	Supero-inferior mm.	Medio-lateral mm.
Right labyrinth on model	34	24	13
Left labyrinth on model	36	28	10
Loeb's average (and range)	35 (22-54)	31.6 (17-59)	16.3 (9-28)

Three groups of cells may be distinguished in the ethmoidal labyrinth: the anterior (designated in the figures by the letter a), the middle (m), and the posterior (p). The anterior and middle groups, which communicate with the middle meatus, are frequently classified together as the anterior group.

The anterior group of ethmoidal cells is painted red in the model. On the right side, four anterior cells (figures 4, 5, a1, a2, a3, a4) are visible. The upper three (a1, a2, a3) are placed anteriorly and laterally to the nasofrontal duct, with which they appear to communicate. The lower cell (a4), which is rudimentary, is attached to the anterior portion of the right infundibulum, lateral to the region of communication between the infundibulum and the nasofrontal duct. On the left side, six anterior ethmoidal cells may be distinguished (figures 3, a1 to a6). As the bone had been fractured in this region, some difficulty was found in determining the exact communications of these cells. However, two cells (a1 and a2) are attached to the anterior aspect of the nasofrontal duct, while another (a3) is attached posteriorly at the junction of the frontal sinus and nasofrontal duct. Just behind these is another (a5) whose communication is uncertain, and lower still another (a6) attached to the infundibulum near its communication with the nasofrontal duct.

The middle or bullar group of ethmoidal cells is painted deep pink in the model. It includes those opening into the middle meatus upon or just above the bulla ethmoidalis. On the right side they are not very well developed, only two (figure 4, m1, m2) appearing just above the depression (b) corresponding to the bulla. On the left side there are three (figure 3, m1, m2, m3). The two lower (m2, m3) apparently correspond to the bulla, with the third (m1) placed just above them.

The posterior group of ethmoidal cells (figures 3, 4, 7) communicate with the superior meatus, and are placed anterior to the sphenoidal sinus of the corresponding side. On the right side there is a large posterior ethmoidal cell, with several diverticula forming more or less distinct secondary cells (figure 4, p1, p2, p3, p4, p5). Below these the superior meatus (sm) presents two indistinct small pouches, evidently representing rudimentary ethmoidal cells of the posterior group. On the left side there are three well developed cells in the posterior group (figure 3, p1, p2, p3). Below these the superior meatus (sm) presents a small outpouching, similar to the rudimentary cells mentioned on the right side.

VI.—THE SPHENOIDAL SINUSES.

The sphenoidal sinuses are visible in the lateral, posterior and superior views of the model (figures 3, 4, 6, 7, ss). They are relatively small (especially the left), as seen by the following table, including the averages (and range) found by Loeb ('09) in thirty sinuses (in fifteen heads):

	Antero-posterior mm.	Supero-inferior mm.	Medio-lateral mm.
Right sphenoidal sinus on model	16	20	18
Left sphenoidal sinus on model..	15	14	12
Loeb's averages (and range)......	21.5 (2-42)	22.8 (4-38)	18.4 (2-35)

Anteriorly the sphenoidal sinuses on the model show the usual proximity to the posterior ethmoidal cells (figures 3, 4, 6, 7, ss) and communicate with the recessus sphenoethmoidalis (seen in figure 7, r). Medially they are separated by a septum, which is comparatively thick except at one point. Inferiorly the sphenoidal sinuses (especially when enlarged) frequently approach the roof of the nasal pharynx. In this case, however, they are widely separated from the pharynx, as seen in lateral and posterior views of the model (figures 3, 4, 6).

The form and size of the sphenoidal sinuses in the model may be compared with the excellent series of casts of the sphenoidal sinuses by Loeb ('12).

VII.—THE NASAL PHARYNX.

The nasal pharynx, on account of its intimate and important relations to the nasal cavity, was included in the model. It is visible in all the views of the model, and calls for but little description.

Anteriorly the nasal pharynx communicates with the nasal fossæ (meatus nasopharyngeus) through the choanæ (figures 3, 4, 6, 8, ch). Each choana measures about twenty millimeters in height by thirteen millimeters in width, which is somewhat smaller than the average size.

Laterally the nasal pharynx presents on the model near the floor and just behind the choana on each side a small prominence corresponding to the ostium pharyngeum of the auditory (eustachian) tube (figures 3, 4, 6, 7, 8, et). Posterior to this is a broad vertical groove, corresponding to the torus tubarius (figures 3, 4, 8, g). Still behind this groove is a vertical ridge, corresponding to the pharyngeal recess, or fossa of Rosenmueller (figures 3, 4, 6, 7, 8, pr). In the posterior view (figure 6) it is evident that in this case the pharyngeal recesses are asymmetric, that on the left side being smaller and placed more inferiorly. (This and other peculiarities on the model were carefully checked up with the original preparation to insure accuracy.)

Inferiorly the nasal pharynx below the pharyngeal recesses becomes narrowed from side to side (figure 6), and especially anteroposteriorly (figures 3, 4), to form the pharyngeal isthmus (pi) which communicates with the oral pharynx.

It is the intention to have this model reproduced and placed on the market, so as to make it accessible to anatomists, rhinologists and others who may find it useful.

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TABLE OF ABBREVIATIONS USED IN THE FIGURES.

a1, a2, a3, a4, a5—Anterior ethmoidal cells.
b—Depression corresponding to bulla ethmoidalis.
c—Communication of maxillary sinus with infundibulum.
c—Choana.
d—Diverticulum of frontal sinus.
et—Ostium pharyngeum of auditory (eustachian) tube.
fs—Frontal sinus.
g—Groove representing the torus tubarius.
i—Infundibulum ethmoidale.
ic—Inferior nasal concha.
im—Inferior nasal meatus.
ls—Lacrimal sac.
m1, m2, m3—Middle ethmoidal cells.
mc—Middle nasal concha.
mm—Middle nasal meatus.
ms—Maxillary sinus.
n—Nasofrontal duct.
N—Naris.
nc—Meatus nasi communis.
nd—Nasolacrimal duct.
nf—Nasal fossa.
np—Nasal pharynx.
p1, p2, p3, p4, p5—Posterior ethmoidal cells.
pi—Pharyngeal isthmus.
pr—Pharyngeal recess.
r—Recessus sphenoethmoidalis.
s—Nasal septum.
sm—Superior nasal meatus.
ss—Sphenoidal sinus.
v—Vestibule.

LEGENDS AND DESCRIPTION OF FIGURES.

FIGURE 1.

Anterior view of the head, to show the general orientation of the model and the surface relations. (X 2/3.)

FIGURE 2.

Lateral view of the head, to show the general orientation of the model and the surface relations. (X 2/3.) For explanation of the parts visible in the model, see Figure 3.

FIGURE 3.

Left lateral view of the model (X 4/5). The nasal fossa (nf) is seen extending upward and backward from the vestibule (v), though largely hidden by the maxillary sinus (ms). Only the anterior and posterior extremities of the inferior meatus (im) and the depression corresponding to the inferior concha (ic) are visible. Above the maxillary sinus may be seen the projection corresponding to the infundibulum (i). Behind the maxillary sinus may be seen the posterior extremities of the middle meatus (mm), the middle concha (mc), and the superior meatus (sm).

Behind the middle and inferior meatuses the nasal cavity is continuous, through the choana (ch) with the nasal portion of the pharynx (np). On the lateral wall of the nasal pharynx a short projection (et) corresponds to the pharyngeal orifice of the auditory (eustachian) tube. Behind this a broad vertical groove (g), corresponding to the torus tubaris, separates the tubal orifice from a thin vertical ridge (pr), representing the pharyngeal recess, or fossa of Rosenmueller. Below this the pharynx is compressed anteroposteriorly in the region of the pharyngeal isthmus (pi).

Above and behind the superior meatus is visible the left sphenoidal sinus (ss). Anterior to this is visible the ethmoidal labyrinth, the cells forming the three groups: posterior (p1, p2, p3), middle (m1, m2, m3), and anterior (a1, a2, a3, a4, a5, a6).

Above the anterior ethmoidal cell group extends the frontal sinus (fs), with a small, cell-like lateral diverticulum (d). The nasofrontal duct is hidden by the anterior ethmoidal cells.

The nasolacrimal duct (nd), in this case abnormally dilated, is seen anterior to the middle meatus, its lower portion being covered by the maxillary sinus. Its continuation upward corresponds to the lacrimal sac (ls), its fundus lying just anterior to the most anterior ethmoidal cell (a2).

FIGURE 4.

Right lateral view of the model (X 4/5). The general relations are similar to those described in Figure 3, with a few exceptions. Of the ethmoidal cells, there are five visible in the posterior group (p1, p2, p3, p4, p5), two in the middle or bullar group (m1, m2), and four in the anterior group (a1, a2, a3, a4). The nasofrontal duct (n) is partly visible behind the anterior ethmoidal cells, where it communicates with the infundibulum (i). Above and behind the infundibulum is the depression corresponding to the bulla (b). The nasolacrimal duct (nd) and lacrimal sac (ls) are not dilated, as on the left side. The communication of the maxillary sinus with the infundibulum is shown at c.

FIGURE 5.

Anterior view of the model (X 4/5). The anterior aspect of the nasal cavity is seen in this view. The interval (s) between the two nasal fossæ (nf) represents the nasal septum which, in this case, shows a marked deviation, with convexity to the right (better shown in the superior view). The vestibule (v) of the right side flares outward. The nares appear at N. On the inferolateral aspect of each nasal fossa is seen the anterior extremity of the inferior meatus (im), lateral to which is the maxillary sinus (ms).

Above the inferior meatus is seen the space corresponding to the inferior concha (ic). Between the inferior concha and the maxillary sinus, on each side, lies the lower portion of the nasolacrimal duct (visible in the figure only on the right side of the model). It unites below with the inferior meatus and extends upward to form the lacrimal sac (ls). Some of the anterior ethmoidal cells (a1, a2, a3, a4) are visible, and above these extends the frontal sinus (fs).

FIGURE 6.

Posterior view of the model (X 4/5). The broad expansion of the nasal pharynx (np) terminates inferiorly at the pharyngeal isthmus (pi). Laterally the extensions formed by the pharyngeal recess (pr), or fossa of Rosenmueller, are visible on each side. Anterior to these the ostium pharyngeum of the auditory (eustachian) tube is visible on the left side only (et). Anteriorly, the nasal pharynx communicates with the nasal fossæ (nf) through the choanæ (ch) on each side of the posterior border of the nasal septum (s). Lateral to the nasal fossæ may be seen the posterior aspects of the superior meatus (sm), superior concha (sc), and middle meatus (mm). The inferior concha and meatus do not appear clearly in the photograph. Lateral to the nasal cavity on each side is the maxillary sinus (ms). More superiorly appear the sphenoidal sinuses (ss), in this case rather small (especially the left) and widely separated. The ethmoidal labyrinth is indistinctly visible. The frontal sinuses (fs) are prominent above, with the beginning of the nasofrontal duct (n) on the right.

FIGURE 7.

Superior view of the model (X 4/5). The deflection of the septum (s), convex to the right, is somewhat exaggerated in this view, which is from above and slightly to the left. This also makes the left nasal fossa (nf) appear wider. The asymmetry of the vestibules (v) is apparent. The left maxillary sinus (ms) appears more prominent, due to the oblique view. Its communication with the middle meatus (at c) is visible on the right side, but concealed by the frontal sinus (fs) on the left. On account of being nearer the photographic lens, the frontal sinuses are out of focus and appear blurred and exaggerated in size. The right nasofrontal duct begins at the point n. The fundus of the distended lacrimal sac (ls) is visible on the left side.

Behind the frontal sinuses the narrow meatus nasi communis (nc) appears on each side of the nasal septum (s). At the posterior extremity of the meatus communis the recessus sphenoethmoidalis (r) is visible. A little of the left middle meatus (mm) is visible. A few of the anterior (a2, a4), middle (m2) and posterior ethmoidal cells (p1, p2, p3) are seen, forming the ethmoidal labyrinth. Behind thees are the sphenoidal cells (ss), the left much smaller, with a rather wide space separating them. Still more posteriorly appears the nasal pharynx (np) with the pharyngeal recesses (pr). The ostium pharyngeum of the auditory (eustachian) tube (et) is visible on the left side only.

FIGURE 8.

Inferior view of the whole (X 4/5). The inferior view is comparatively simple, made up chiefly of the floor of the inferior meatus of the nasal fossæ (nf) on each side of the nasal septum (s). Anteriorly are the vestibules (v) and nares (N). Posteriorly is the communication through the choanæ (ch) with the nasal pharynx (np). This presents the pharyngeal ostia of the auditory (eustachian) tube (et). Behind these on each side is a groove (g), corresponding to the torus tubarius, next to which is a lateral extension (pr) on each side, corresponding to the pharyngeal recess (fossa of Rosenmueller). The posterior extremity of the model corresponds to the pharyngeal isthmus (pi). Lateral to the nasal cavity on each side appears the floor of the maxillary sinus (ms).

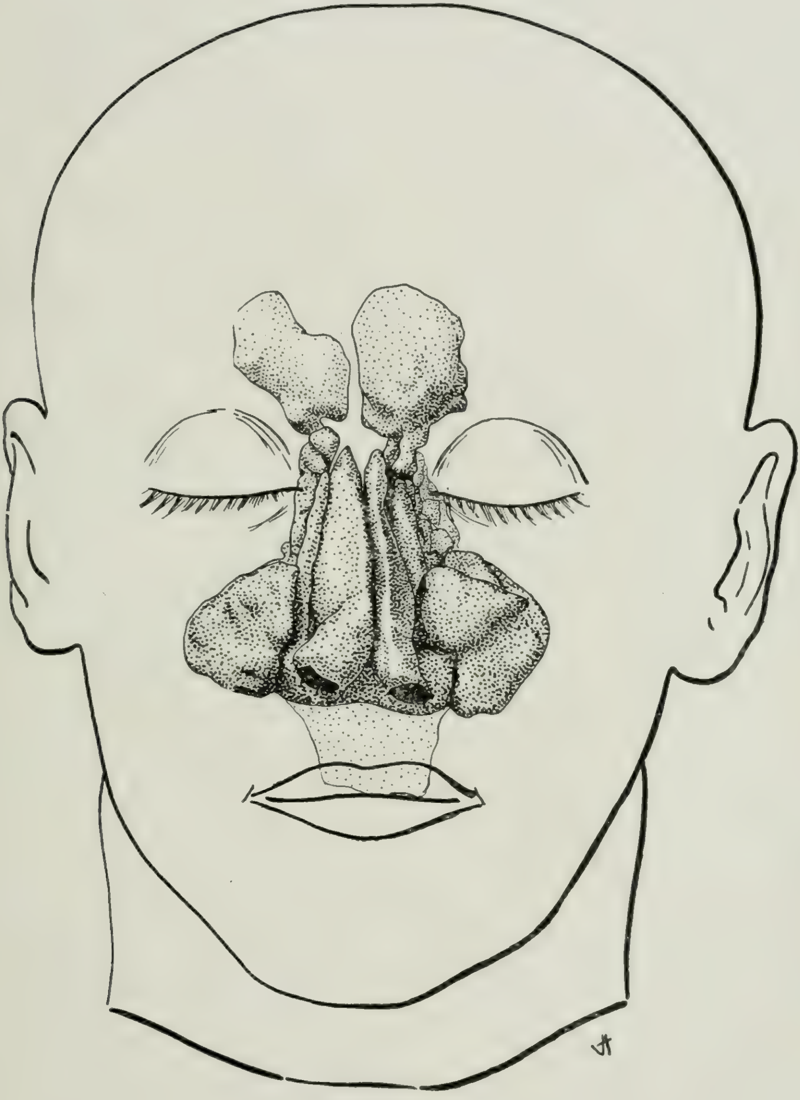

FIGURE 1.

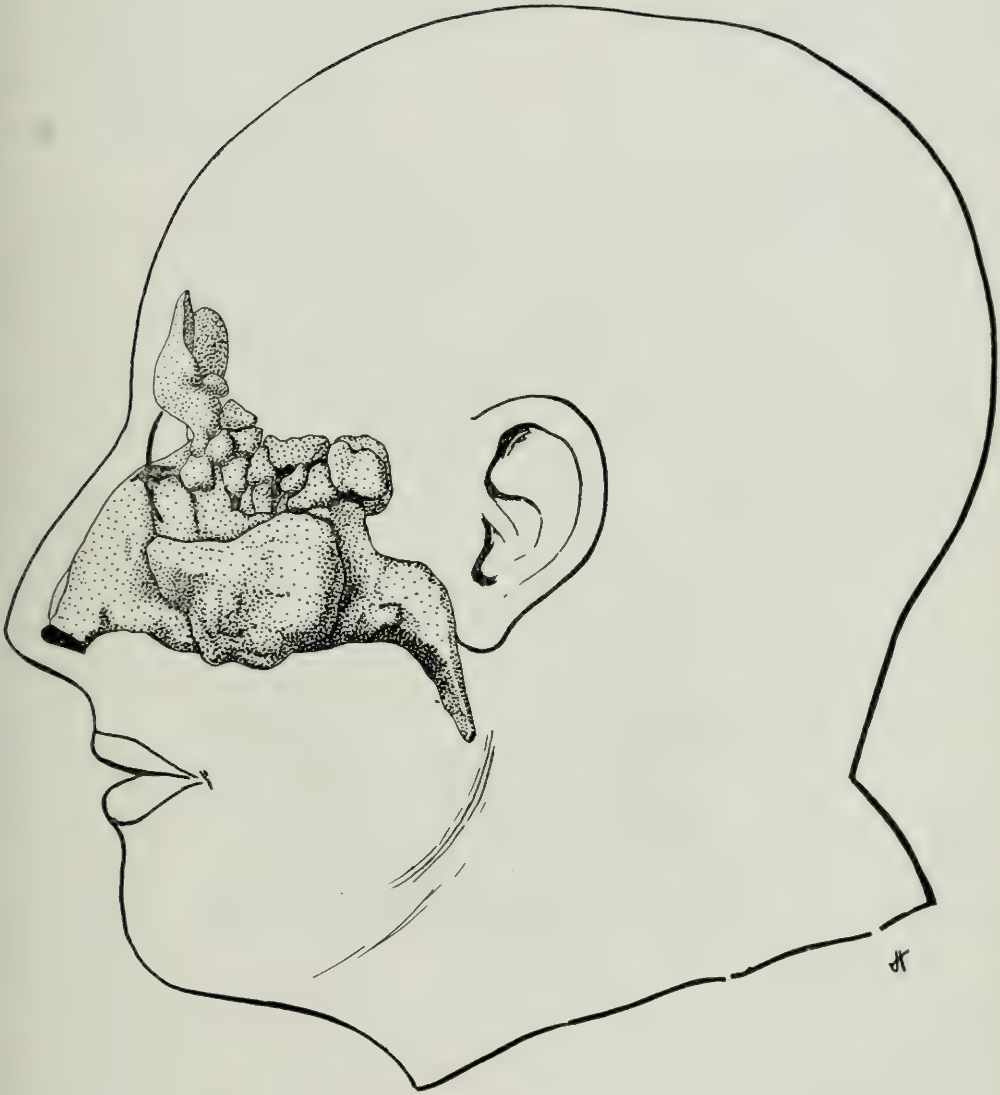

FIGURE 2.

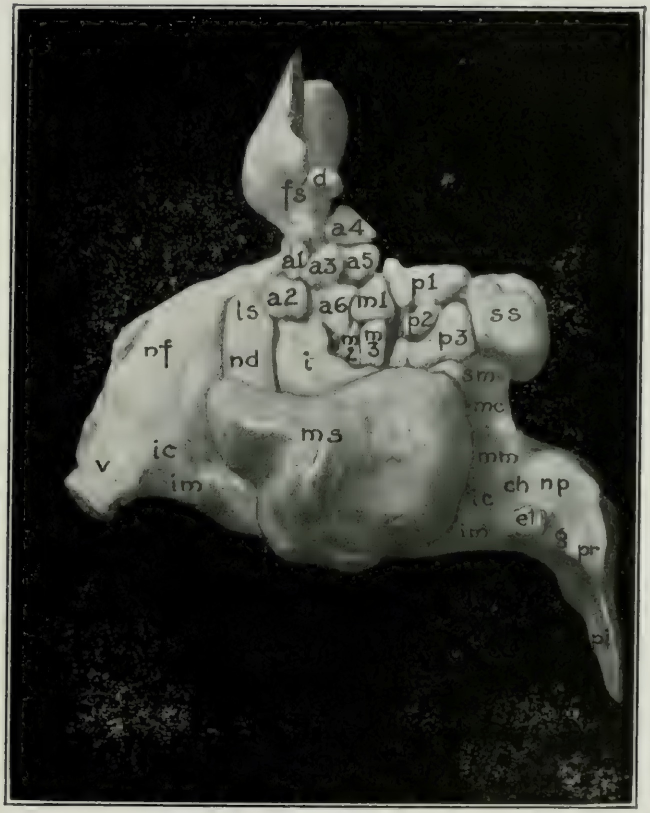
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FIGURE 3.

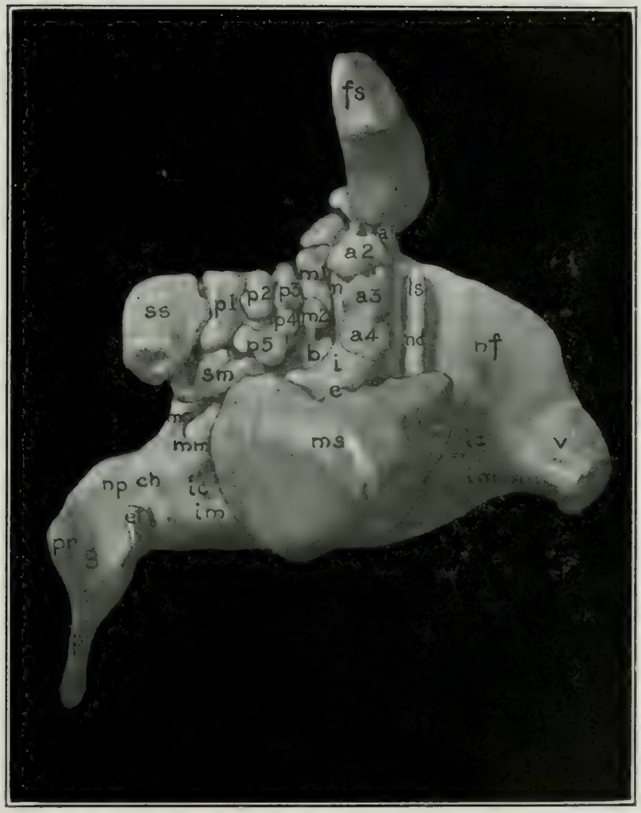
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FIGURE 4.

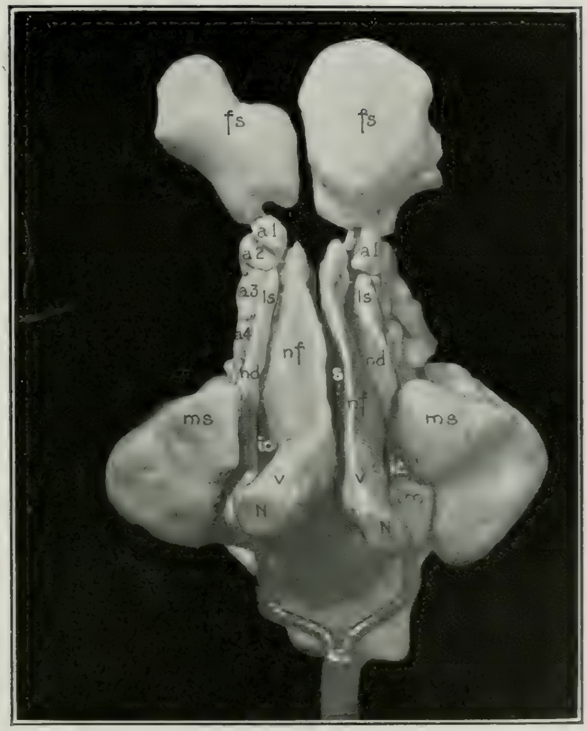

FIGURE 5.

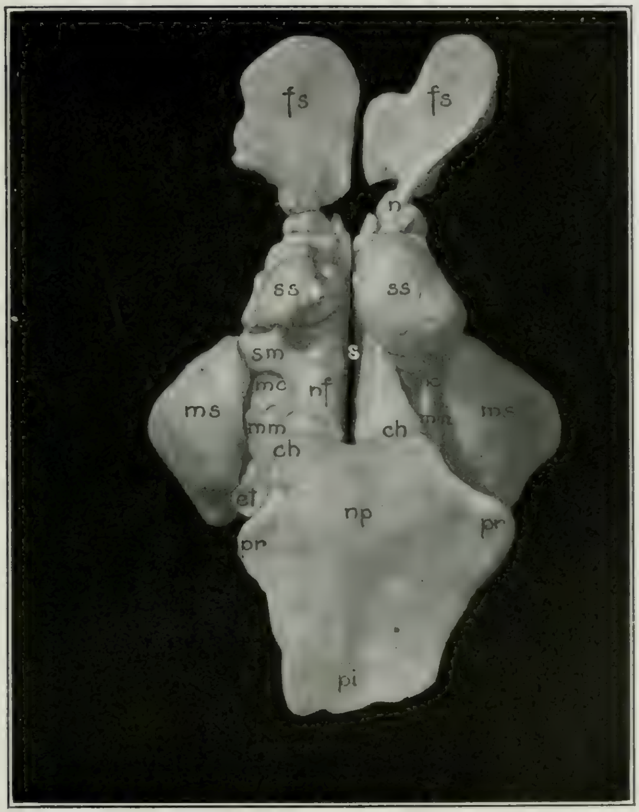

FIGURE 6.

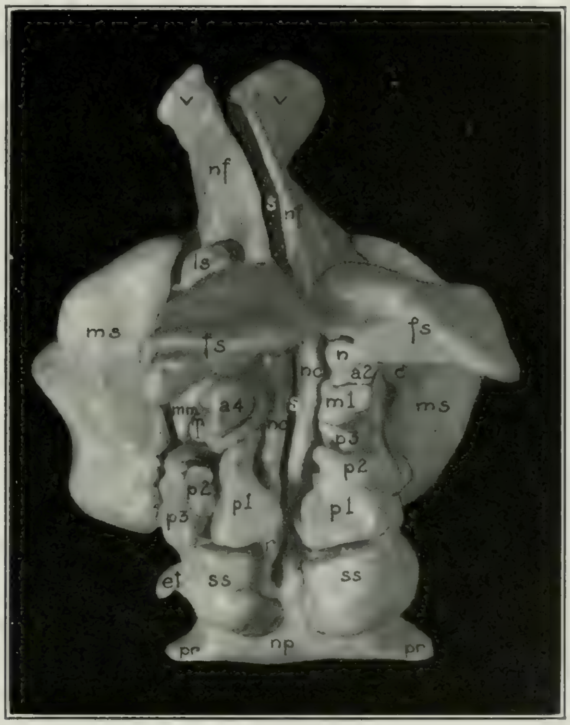
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FIGURE 7.

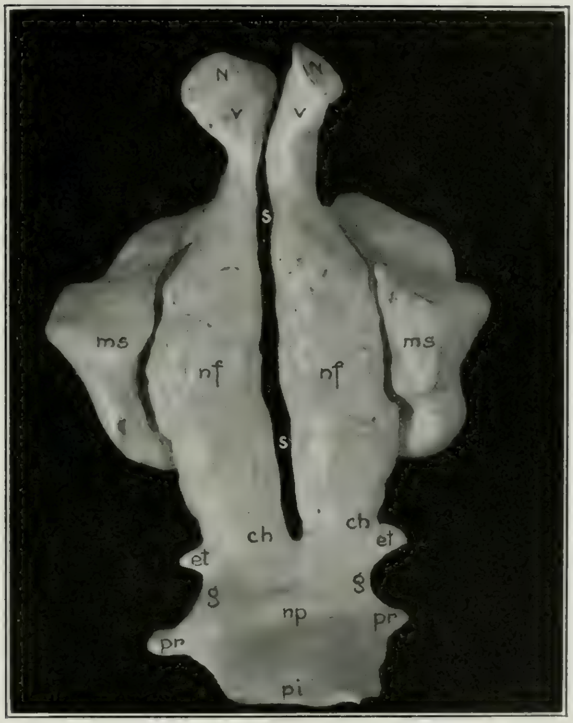

FIGURE 8.

XXXVII.

CLOSURE OF OBSTINATE OPENINGS OF THE DRUMHEAD.

By Frank Allport, M. D.,

Chicago.

A few times in my life I have been confronted with an incorrigible drumhead opening. The literature on the subject is not helpful nor extensive, and yet these cases are sometimes of very great importance. They may involve hearing and chronic discharge or litigation, or all three, to say nothing of impaired reputation. I would suggest, therefore, that anyone having an experience of a helpful nature in such cases should place it upon record that others may be benefited. Acting upon this suggestion, I beg to report the following case:

Mrs. M. E., an old friend and patient (I mention the fact that she is an old friend, because I am quite confident that had not this been the case she would have wandered into other hands, and I should not have blamed her if she had) consulted me on January 15, 1917, with an abscess of the left middle ear that had not yet broken through the tympanic membrane. She was suffering much pain, and I made the usual horseshoe incision through the drumhead. Her suffering ceased and the discharge soon dried up. She was left, however, with a good-sized opening in the lower portion of the drumhead. Her tympanic cavity was free from discharge. Her hearing was very poor, and this was all the more regrettable because she had been almost completely deaf in her other, or right ear, for many years. She was thus dependent on her hearing in the left ear. Every effort was made to close this up. I used nitrate of silver in varying strengths. Insufflations of powdered boracic acid were tried. I scarified the edges of the opening. I used various kinds of artificial ear drums for protection, such as cotton cones, Blake's taper discs over the opening, the discs being previously lightly dipped in alcohol. I used tincture of iodin and various other remedies, but all to no avail. About May 1, 1917, I read an article in *Laryngoscope,* by Dr. A. L. Dunlap of Shanghai, China, on the Okuneff trichloracetic acid method of treating this character of cases. I immediately put it into practice, and having previously applied a ten per cent solution of cocain to the parts, applied with a small cotton probe tightly wrapped with very little cotton, a small amount of the trichloracetic acid, limiting this application, as far as possible, to the edges of the perforation. It is not possible to confine the application exclusively to the edges of the perforation, but an effort should be made to do this as nearly as possible. The opening was then protected by a small cone of tightly twisted cotton. The opening soon diminished in size. Three applications were made at intervals of about a week. After the last application a scab formed over the opening, which was removed after a few days, and the patient was dismissed as cured with perfect hearing, May 25, 1917.

XXXVIII.

REPORT OF A CASE OF COEXISTENT CARCINOMA, TUBERCULOSIS, AND SYPHILIS OF THE ESOPHAGUS.

By L. W. Dean, M. D., and J. B. Gregg, M. D.,

Iowa City, Iowa.

The purpose of this article shall be to briefly discuss carcinoma, tuberculosis, and syphilis of the esophagus; their coexistence in this organ, with the report of a very unusual case.

TUBERCULOSIS OF THE ESOPHAGUS.

The first case of esophageal tuberculosis was recorded by Denonvilliers,[1] 1837, and six years later Louis reported finding six cases of esophageal ulceration, presumably tuberculous, in one hundred and twenty tuberculous cadavers.

In the seventy-four years which followed this first observation, one hundred and three cases were reported; only sixty-nine of which can, with reasonable certainty, be classed as tuberculous. Since then numerous cases have been reported, until now the affection is no longer regarded as being of great rarity.

This disease has, however, received such slight consideration in medical literature that the physician does not take it into consideration in an otherwise tuberculous patient, and may ascribe the esophageal symptoms to a visible laryngeal tuberculosis.

The classification according to etiology, as suggested by Flexner,[2] is as follows:

1. Instances in which the tuberculous process arises through continuity or contiguity of structure; as in ulceration of the esophagus from a caseous bronchial gland, caries of the vertebræ, or from a tuberculous ulcer of the pharynx which passes down and invades the esophagus.

2. Cases in which there exists in the esophageal mucosa a previous lesion to be regarded as predisposing to tuberculous infection.

3. Instances in which (a) the esophagus is affected in the course of a general disseminated miliary tuberculosis; and (b) in which there is infection of the mucosa from tuberculous sputum where no previous lesion existed.

Of the nineteen cases of undoubted tuberculosis collected by Flexner, but two cases belonged to Class 2, in which we are especially interested in this report.

When seen as an extension from the larynx, especially as "party wall" lesions, the esophageal lesions present much the same picture as the laryngeal disease.[3] When, however, they are not confluent with a lesion in the pharynx or larynx in the esophagus, the endoscopic appearances are those of superficial ulceration, or a simple erosion, and there may be yellowish or whitish granules in the neighborhood of the erosions or ulceration. Cicatrices have been observed by Jackson but twice: once in connection with ulceration, and once in invasion of the esophagus by peribronchial tuberculous glandular processes. The ulcerations of esophageal tuberculosis usually are more superficial and less inflammatory in character than either cancer or lues. The diagnosis here, as elsewhere, rests upon the microscopic findings in pieces of involved tissue removed by means of the esophagoscope.

SYPHILIS OF THE ESOPHAGUS.

Syphilis involving the esophagus, although rare, has long been known to exist, having been described by Marcus Aurelio Severino (1580-1656).[4] Prior to the days of esophagoscopy,[5] the few diagnoses of lues of the esophagus were made solely from the disappearance of esophageal symptoms following antiluetic treatment. Such diagnoses necessarily are apt to err.[6]

The esophagoscope has shown that syphilis of the esophagus may present itself as the mucous plaque, a gumma, an ulcer, or as a cicatrix. For diagnosis, the esophagoscopic appearance must be considered in relation to the history, the concomitant lesions, the therapeutic test, the Wassermann reaction, the examination of the tissue, and the search for spirochetes.

The esophagoscopic appearance of the luetic ulcer is largely due to mixed infection, with resultant inflammatory condition. The mucous plaque appears similar to that seen in the fauces. It may be slightly elevated in one part, with simply a bluish white cloudiness in another part. The lesion is typically inflammatory in character. The cicatrix of luetic esophagitis does not differ from other cicatrices.[7]

CARCINOMA OF THE ESOPHAGUS.

Carcinoma is the most important neoplasm and the most common disease affecting the esophagus.

Eighty per cent of malignant esophageal cases are carcinomatous.[8]

In 5079 autopsies, Zenker[9] found carcinoma of the esophagus primary in only thirteen cases, and secondary in but six. Williams[10] has found them to constitute 5.3 per cent of all carcinomatous tumors, and to be much more frequent in men than in women. Von Hacker[11] states that over seventy-two per cent occur in males, and that the neoplasm is usually primary in the esophagus. They may develop by extension or by implantation,[12] but do not occur in this locality as the result of metastasis.[13]

Carcinoma of neighboring organs may spread to the esophagus and be inoculated from a cancer situated higher up or lower down, and the entire tube may become covered with carcinomatous nodules. The cancer is usually an epithelioma (pavement epithelium) ; glandular cancer and the colloid form are more rare. As a rule, only a portion of the esophagus is attacked, with the production of insular infiltration forming a segment on one side of the tube, or annular, cauliflower-like and papillomatous tumors.

Carcinoma of the esophagus is found most frequently in one of three localities : at the cardiac orifice, opposite the bifurcation of the trachea, or on a level with the cricoid cartilage.

Jackson gives the following forms of esophageal carcinoma :

1. Submucosal infiltration covered by perfectly normal membrane ; usually associated with more or less bulging of the esophageal wall, and usually associated with hardness and infiltration.
2. Leucoplakia.

3. Ulceration projecting but little above the surface at the edges.
4. Rounded nodular masses grouped in mulberry-like form, either dark or light in color.
5. Polypoid masses.
6. Cauliflower fungations.

It is always well to remember that a carcinomatous lesion which has existed for some time may have engrafted upon it a mixed infection, with resutling inflammation.

The real diagnosis rests on the histologic examination of a specimen removed esophagoscopically. Submucous tumors should not have a piece removed for diagnosis because of the danger of causing ulceration and rapidly increasing the growth of the tumor if it be malignant.

Seventy per cent of the cases of esophageal carcinoma have been found in patients above fifty years, but it has also been observed in young individuals (nineteen years of age).

Irritation, mechanical, chemical or thermal, is a most important predisposing factor in the etiology.[14]

Zenker believes that alcohol is a very common irritant to the esophageal mucosa, and may predispose to carcinoma.

Körner[15] and Voelker attribute most esophageal carcinomata to the irritation produced by the passage of large boluses of poorly masticated food, and this has been in accordance with the apparent etiology of the cases with which we have dealt.

It is doubtful if any antagonism exists between carcinoma and tuberculosis; there is also no special favoritism; yet cancer is more frequent among those with latent tuberculosis than among those at the cancer period of life.[16]

The coexistence of tuberculous disease and cancer in the same part has been shown by Crawford, in the mammæ; Franque, in the uterus; Frerichs, in the liver; Beale,[17] in the larynx; Dalton, in the large intestine; Naeghi, in the rectum, ilium and cæcum; Dixon,[18] in the intestine; and by Lapeyre,[19] in the hand.

Certain observers have held that the tubercle bacillus and its toxins are concerned in the etiology of cancer, but Dixon, Smith and Fox,[20] after animal experimentation, convinced themselves that the tubercle bacillus and its products could stimulate epithelial structures to hyperplasia, but not produce an invasive tumor. To explain the combination of the two diseases two theories have been suggested:[21]

First, that the chronic inflammatory action of tuberculosis prepared the way for the development of cancer.

Second, that cancer, by lowering the general nutrition, assisted the growth of tuberculosis.

Cases of coincident carcinoma and tuberculosis of the esophagus have been reported.[22]

Zenker[23] reports a case of stricture of the tube produced by a girdle ulcer of rodent character, which on examination microscopically proved to have the typical appearances of a squamous epithelioma. The growth invaded the deeper strata of the mucosa, and at times penetrated the muscularis. In the border of the cancerous portion, near the normal tissue, typical tubercles existed, some of which were caseous. The neighboring lymph glands were also involved in the cancerous and tuberculous conditions. Tubercle bacilli were found in small numbers in the tubercles. Zenker favors the view that the carcinomatous lesion was primary, the intact epithelium being resultant to tuberculous infection.

Cordna[24] reports the case of a man of sixty years, who came to autopsy with the clinical diagnosis of carcinoma of the esophagus, incipient pulmonary tuberculosis, and adhesive pleuritis. In the esophagus, at about the bifurcation of the trachea, was found an ulcer ten to fifteen centimeters long, which affected the whole circumference. The edges of the ulcer were somewhat elevated and firm, the base was soft and pulpy. Upon sectioning the ulcer, the entire wall of the esophagus was found almost destroyed, forming a small tumor mass. Microscopically, both a carcinomatous and a tuberculous lesion was found. Tubercles in the process of caseation were found in the middle of the cancerous ulcer and about its periphery. No tubercle bacilli were found after careful search.

Cordna thought the tuberculous invasion of the cancerous ulcer took place through the lymphatics leading from tuberculous areas in the lungs. It would seem, however, more probable that the secondary tuberculous involvement was due to sputum deglutition.

Pepper and Edsall[25] [26] report the case of a man, forty-two years of age, who had tuberculosis of the lungs, larynx, cervical glands, and esophagus. The esophagus was patulous and normal as high up as the level of the arch of the aorta. Above this point and to the junction with the pharynx, the lumen had entirely disappeared.

The esophageal walls and surrounding tissues had formed a band of dense fibrous tissue about 1.25 centimeters thick and 3.75 centimeters broad, which was firmly bound to the vertebræ. Examined microscopically, cancerous tissue was found on a dense fibrous tissue basis; a tubercle-like mass undergoing fibroid changes, with an occasional giant cell, and tubercle bacilli.

The central and deeper portions of the tumor were composed mainly of fibrous tissue; this leading the authors to conclude that tuberculosis was the original cause of the disease, while cancer subsequently infiltrated the occlusion.

Weyeneth[27] reports the case of a man of fifty-eight years, who had pulmonary tuberculosis for five years, for four years suppuration and deafness of the left ear; and later involvement of the right ear. At the time of examination there had recently developed difficult deglutition and vomiting after eating. A sound was passed into the stomach without meeting an obstruction. After three weeks death occurred, the cause being given as "tuberculosis pulmonum et intestini." The autopsy showed tuberculosis of both lungs and several ulcers in the neighborhood of the ileocecal valve. On the anterior wall of the esophagus, and on a level with the bifurcation of the trachea, was an oval ulcer that macroscopically gave the impression of an ulcerated tumor. Surrounding the ulcer was an area over which the mucosa was destroyed. Scattered throughout this denuded area were numerous small nodules, both isolated and grouped. Granulation tissue covered the base of the ulcer, and the esophageal wall in the neighborhood of the ulcer was thickened. Microscopically the chief mass was found to be composed of cancer tissue with recent tuberculosis, tubercle bacilli being found in the nodules about the periphery.

In this case the sequence was: tuberculosis of the lungs and intestines; carcinoma of the esophagus; tuberculosis of the esophagus. Weyeneth's opinion, concurred in by Professor Ribbert, was that the malignant growth antedated by several months the tuberculous invasion. Infection by the sputum[28] seems probable, as there were no foci in the neighboring lymph glands.

Syphilis in combination with carcinoma or with tuberculosis is more rare.

Kellert[29] has found relatively few cancer patients who gave a specific history, and Wassermann tests on their blood serum seldom gave a positive reaction.

Occasionally[8] leucoplakia has been observed in the esophagus, similar to that of other mucous membranes, coincident with carcinoma of the esophagus. As carcinoma develops in other situations where there is leucoplakia, it is possible that here also it may occur in some cases of syphilis as the causal agent for the leucoplakia.

According to Lexer Bevan,[30] carcinomas of the mucous membranes may develop from fibroepithelial growths and from ulcers of all kinds, especially the tuberculous and syphilitic.

Dennis[31] has notes on a case in which a diagnosis of tuberculous laryngitis was made in a woman with a history of pulmonary and hip joint tuberculosis. Later a characteristic skin syphilid was discovered, and under antiluetic treatment the larynx rapidly healed, except in a comparatively small area, which remained obstinate. He considered that this was probably a case of syphilis of the larynx on which a tuberculous infection was grafted.

According to Dennis,[31] Moritz Schmidt has reported a case in which tuberculosis, syphilis and carcinoma were all present in the larynx of the same patient.

Other writers[32] [33] report laryngeal cancer having coexisted with laryngeal syphilis and with laryngeal tuberculosis.

Jackson[34] reported a case before this society, in which he was certain that carcinoma, syphilis and tuberculosis coexisted in the larynx. The patient, a man of fifty-two years, had a family history of tuberculosis, a history of a primary luetic lesion three years before, followed by well marked secondaries, and at first examination a gumma of the septum. The larynx was in the ulcerative stage, the respiratory chink being narrowed to an oblique slot. Fungating masses protruded from the ulcers, but not sufficiently to completely occlude the laryngeal lumen. The neck was full of enlarged hard lymphatic glands. Jackson considered the diagnosis of laryngeal syphilis with perichondritis indisputable, and the patient was put on vigorous mixed treatment, with marked laryngeal improvement. Healing occurred until the only lesion was a fungous, warty-looking mass on the left cord and filling the left ventricle. Here all improvement ceased, and overwhelming doses of potassium iodid, with mercury to full tolerance, for two months, had no effect on the growth, which began slowly to increase in size. Repeated examinations of the sputum revealed tubercle bacilli. A specimen of the larynx was removed and proven to be an epithelioma.

Kellert[29] reports the case of a woman whose postmortem examination led to the following anatomic diagnosis:

Carcinoma of the pharynx with metastases to the cervical lymph node; tuberculosis involving the same area of the pharynx; tuberculosis of the same lymph nodes as involved by the carcinoma; pulmonary tuberculosis with cavity formation; tuberculous pleuritis with effusions; tuberculous ulcers of the small intestine; and tabes dorsalis, syphilitic.

Moritz Schmidt[35] has observed a case with Roth, which was diagnosed tuberculosis of the larynx. There was healing of this lesion, yet in spite of the fact that the patient denied syphilis, tertiary lesions were found in the larynx. There was also partial healing of this second lesion, but a growth diagnosed microscopically by Weigirt as carcinoma, appeared upon this site, and from which the patient finally died.

Case.—Mr. J. S., white, male, sixty years of age, German, a tailor. Referred by Doctor F. W. Bailey of Cedar Rapids, Iowa.

The patient entered the University Hospital, April 17, 1916, complaining of inability to swallow well, weakness and cough.

Present Illness.—Twenty-eight years ago he first had trouble in swallowing, lasting for five weeks. His only trouble at that time was dysphagia; no loss of weight, no cough, no regurgitation or lodging of food. This attack began suddenly and ended suddenly without treatment of any kind. Since that time up to November, 1915, he had no trouble of any kind.

In November, 1915, while eating supper, he swallowed a piece of meat not well masticated, which seemed to lodge just back of the sternum, causing violent coughing. The coughing spell soon ended and the feeling as of something lodged in the throat disappeared. He drank some liquids at once without difficulty, and the next morning he ate as usual.

For about six weeks he swallowed as well as ever, but about the last of December, 1915, he noticed that he was becoming poor. About January 1, 1916, he began again to have some trouble in swallowing, the amount of dysphagia varying considerably. Gradually the swallowing became more difficult, so that articles of solid food were not eaten. Even in swallowing soft food he would often choke and cough so violently that food was expelled through both the nose and the mouth. He coughed a good deal, but not at any particular time; no event seemed to bring on these attacks. No blood was ever coughed up; only a very tenacious slimy sputum, like the white of an egg, only more sticky. He does not regurgitate food eaten hours before. The throat feels dry, and he has marked thirst, which, owing to the difficulty in swallowing, he cannot satisfy.

At the present time he is unable to swallow anything with surety. He has lost fifty pounds in weight, and is markedly weakened, sallow and anemic. He complains of pain in the chest just behind the sternum, which is severe enough to awaken him at night. During these attacks of pain he finds it difficult to breathe.

Family History.—His father and mother are dead, the cause of death being unknown. He has five half brothers alive and well; one half sister alive and well.

He has four children alive and well. His wife has had three miscarriages, and one stillbirth.

Past History.—He had measles as a child, but no other childhood diseases; no fevers with the exception of malaria.

He uses beer and whisky moderately, never to excess. He chews tobacco moderately. He denies venereal history, but in 1887 he had a swelling of the cervical glands and a very sore throat. No other glands became enlarged, and there was no skin eruption. He denies sweats and hemoptysis; has had some cough, but not excessive. He is susceptible to cold, but there has never been any dyspnea.

He was hit on the head at one time, resulting in a skull fracture which caused the left ear to become deaf.

General examination by Doctor C. P. Howard, May 5, 1916. The patient is a poorly nourished and developed man of stated age, and gives the appearance of having lost much weight. The temporal arteries stand out prominently.

The muscles are small, and there is a marked myoidema. The skin is hot, moist and covered with acne, without scars or areas of pigmentation suggesting a previous lues.

The thorax is of the senile emphysematous type, with high clavicles and rounded back. Expansion is fairly restricted equally. Litten is present on both sides. The percussion note is definitely impaired in the right supraspinous fossa and over the right clavicle. No changes in vocal fremitus. The breath sounds are feeble and vesicular, nowhere prolonged. Definite fine crepitations are heard over the apex, spine, clavicle and scapula on the right side, an occasional crepitation being heard over the left scapula.

Heart.—The point of maximal intensity is in the fifth interspace eight centimeters from the midsternal line. The area of relative cardiac dullness extends four centimeters to the right of the midsternal line and seven and one-half centimeters to the left. The heart sounds are distant; no murmurs; a_2 is not definitely accentuated.

The radial pulse is synchronous and equal; of good volume.

The blood pressure is: systolic, 118 millimeters Hg.; diastolic, 60 millimeters Hg.

There is no tracheal tug and no retrosternal dullness.

The abdomen is flat and doughy; no masses or areas of dullness. The liver or spleen cannot be felt.

The shins are free from edema. There are two or three nodes over the right shin; possibly traumatic. The reflexes are normal on each side.

Diagnosis.—Tuberculous involvement of both apices.

ROUTINE EXAMINATION.

Eyes.—Chronic catarrhal conjunctivitis is present, with some lacrimation; tear ducts patent. The pupils are round, equal, two millimeters in diameter, reacting to light and accommodation. Rotations are normal. The media are clear; the fundi showing no evident lesion.

Right eye, vision equals 6/60. Left eye, vision equals 6/60.

Ears.—Right. The canal is clean; drum retracted slightly, but the ossicles move with Siegel's otoscope. High notes are well heard, but low notes are slightly cut down. He hears A, C^3 and C^4 tuning forks; not the A fork. Weber localized to the right.

Left. The canal is clean, drum retracted, the ossicles not moving with Siegel's otoscope. Galton whistle and none of the tuning forks are heard by air.

Hearing with alarm.—Right, whispered voice, twelve feet; spoken voice, forty-five feet. Left, whispered voice, 0; spoken voice, 0.

Nose.—The septum is deflected to the right, with a large spur on the left. Each middle turbinate is enlarged. There is a small amount of mucopus in each middle meatus.

Throat.—The teeth are in very poor condition, with marked pyorrhea. The pharyngeal mucosa and posterior buccal mucosa are markedly reddened. The lower pharynx and larynx are covered by a profuse amount of mucus.

Esophageal Examination.—On swallowing liquids he coughs and chokes, nearly all the fluid being ejected. No fluid can be heard entering the stomach, and there is no gurgling in the neck. Choking comes immediately after swallowing.

The radiogram taken after swallowing bismuth and acacia mixture shows an S-shaped stricture of the esophagus; the convexity of the upper portion is to the right, of the lower portion, to the left. The middle of the stricture is at about the union of the manubrioclavicular articulation. The stricture apparently begins at the level of the lower margin of the cricoid cartilage.

April 21, 1916, the patient was esophagoscoped under cocain anesthesia, using the Mosher and Jackson esophagoscopes.

One and one-half inches below the cricoid cartilage was found a nodular mass on the right side of the esophagus. The lumen of the tube was very small and to the left of the mass. Pieces of tissue were removed from this mass with biters, for pathologic examination.

Carcinoma and tuberculosis of the esophagus.

Report of Doctor Albert on tissue removed from esophagus:

Gross Pathology.—The material to be examined consisted of tissue scrapings, the mass of which measured about one centimeter in diameter, and individual pieces of which measured from two five millimeters in diameter.

Microscopic Pathology.—Certain pieces of tissue consisted of the normal squamous epithelial lining of the esophagus. Others, and more especially the largest piece, consisted of connective tissue and nonstriated muscle tissue invaded by a large number of solid masses of epithelial cells. These epithelial cells were of the squamous type. Many of the nests or masses of epithelial cells contained hornified epithelium in their centers, forming distinct whorls or pearls. The muscle or connective tissue invaded by the epithelial cell masses is markedly inflamed, as shown by a large amount of "round cell" infiltration, the round cells of which are principally fibroblasts.

Other pieces of tissue contained distinct tubercles consisting of central masses of beginning caseation necrosis surrounded by quite a large number of giant and endothelial cells, and about the periphery, large number of round cells consisting of fibroblasts and lymphocytes. The giant cells were of the typical tubercle type—that is, the nuclei were arranged about the periphery of the cells, whereas the center of the cells showed the homogeneous structure of caseation necrosis. In one field, fourteen giant cells were found. A few tubercle bacilli were also found. (Plate 986, Plate 986a.)

April 22, 1916, the Wassermann test on the blood was positive.

May 7, 1916, the tuberculin test (von Pirquet) was positive.

May 2, 1916, tubercle bacilli were found in the sputum by Doctor Royce.

May 15, 1916, smears taken directly from the larynx were reported negative for tubercle bacilli by Doctor Royce.

The blood examination on May 7, 1916, revealed the following: Hemoglobin, seventy-five per cent Sahli. Red cells, 4,900,000; white cells, 12,600. Differential count: Polymorphonuclears, seventy-one per cent; lymphocytes, twenty-one per cent; transitionals, two per cent; large mononuclears, four per cent; eosinophiles, one per cent; basophiles, one per cent.

The urine was yellow; specific gravity, 1008; acid; negative for albumin, sugar and sediment. Microscopically a few hyaline casts were found.

The patient was placed on antiluetic treatment April 24, 1916; mercury inunctions and potassium iodid. A marked improvement in dysphagia was immediately noticed.

By May 15, 1916, he was able to swallow all kinds of soft food and semisolids. He ate meat, potatoes, vegetables, and had no pain or difficulty while swallowing or afterwards.

Microscopic examination of the pieces removed from the esophageal tumor proved both its carcinomatous and tuberculous character. The marked improvement in the swallowing after antiluetic treatment alone, and in the presence of a positive Wassermann, suggests a definite luetic element present in this mixed tumor. Difficulty in swallowing twenty years ago, which lasted six weeks and then disappeared, is also suggestive of syphilis.

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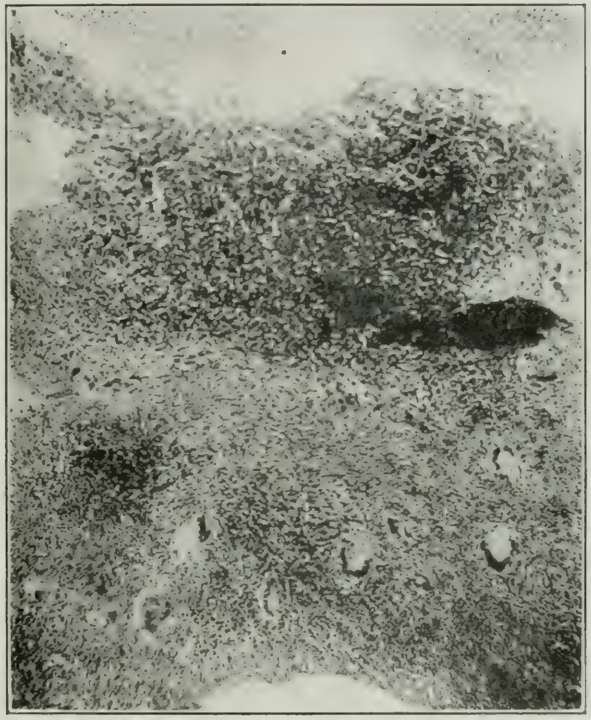

FIGURE 1.

Tuberculous portion of case of carcinoma and tuberculosis of the esophagus. (Plate No. 986.)

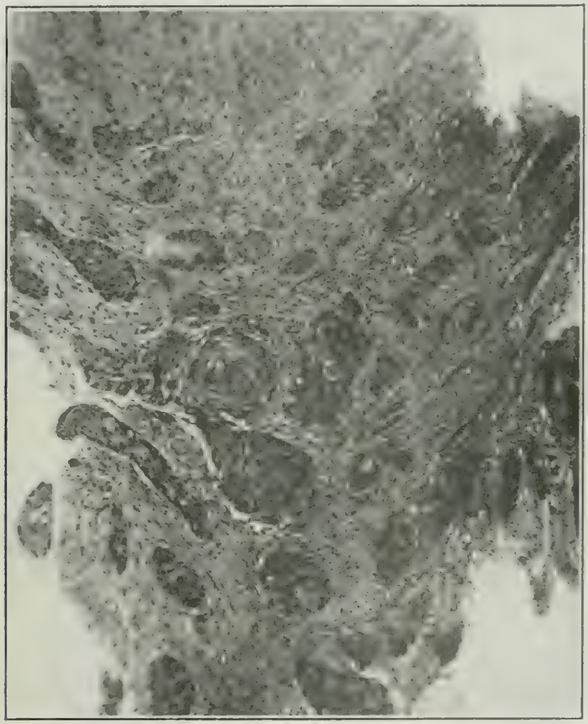

FIGURE 2.

Carcinomatous portion of case of carcinoma and tuberculosis of the esophagus. (Plate No. 986a.)

XXXIX.

STEREOSCOPIC ROENTGENOGRAMS OF THE HEAD.

By J. M. Ingersoll, A. M., M. D.,

Cleveland.

THE TECHNIC FOR MAKING STEREOSCOPIC ROENTGENOGRAMS OF THE HEAD.

BY

W. C. Hill, A. B., M. D., and G. F. Thomas, A. B., M. D.,

Cleveland.

During the past five years we have had stereoscopic roentgenograms of the head made in all cases in which we have done any major operations upon the head. The longer we have used the stereoroentgenograms the more certainly have we been convinced of their practical value, for they give us definite information in regard to the nose and the nasal accessory sinuses, the brain and many of its blood vessels, the ear and the mastoid, which cannot be obtained in any other way. The size and boundaries of the maxillary and frontal sinuses can be distinctly seen. If there are any septa, tumors or foreign bodies in these cavities, they can be accurately located and defined. The ethmoidal and sphenoidal sinuses overlie each other, and are thus somewhat masked, but their position, relative to each other and the orbit and the other surrounding structures, can be clearly distinguished.

The location of malignant tumors and the amount of involvement of the structures by such tumors in the ethmoidal and sphenoidal sinuses is a little less definite than it is in the frontal and maxillary sinuses, but in all cases it is much more definite and accurate in the stereoscopic plates than it is in a flat plate. Purulent infections in the sinuses usually cast a heavier shadow than the normal structures, and so are easily recognized, but occasionally a sinus with pus in it seems to transmit the roentgen rays nearly as well as an uninfected sinus.

Dimpling, or little depressions in the inner table of the skull, indicates increased intracranial pressure, and is almost positive evidence of brain tumor or abscess or hydrocephalus, except in microcephalic skulls. In such cases the dimpling is usually very evident and is due to the increased intracranial pressure within the abnormally small skull. The depth and extent of the depressions usually give some idea of the length of time that the increased intracranial pressure has been present.

The density or specific gravity of the majority of brain tumors and brain abscesses is about the same as the brain tissue, and so they do not cast any shadow in the roentgenogram. If the tumors are dense or calcified, they can be accurately located. If a brain abscess is encapsulated, its shadow will usually show in the plate, and in some cases the abscess may be very definitely outlined.

Skill in interpreting the roentgenograms can be more easily acquired by the surgeon than by the roentgenologist, for the surgeon has the decided advantage of being able to verify and correct the findings in the roentgenogram while he is operating. If the surgeon will carefully study the stereoscopic picture before operating, compare his interpretation of the picture with the condition which he finds in the operation, and then study the picture again after the operation, he will soon acquire great skill in interpreting the stereoscopic plates.

The study of stereoscopic plates of a macerated skull shows distinctly all the principal anatomic points, and enables one, otherwise unfamiliar with stereoscopic plates, to recognize easily the landmarks in the picture of the living subject. In studying stereoscopic plates they should be examined from both sides. First, put the plates in the stereoscope with the film sides toward the mirrors. This will show the structures as we see them in the operation, with the external parts in the foreground and the deeper structures in the background. Then by reversing the plates in the stereoscope, turning the smooth sides of the plates toward the mirrors, the structures will be seen from the inside of the skull.

I have with me for demonstration in the stereoscope the following plates:

Macerated skull, anterior-posterior view, showing all the accessory cavities of the nose.

Plates showing both mastoids of this skull at the same time. Copper wires have been placed in the semicircular canals of the right labyrinth so as to identify them positively. The advantage of taking both mastoids on one plate is selfevident. If only one mastoid is infected, the uninfected mastoid serves as a normal standard, so that both the normal and the infected mastoid can be studied and compared at the same time.

Plates showing an osteoma which developed in the left frontal sinus and extended into the right frontal sinus, both orbits, both nasal fossæ and the brain cavity, in spite of an operation during the early development of the growth, in which I thought I had removed all the tumor.

Plates showing a bullet lodged in the right mastoid antrum after it had passed through the left orbit and posterior ethmoidal cells, the right sphenoidal cavity and temporal lobe of the brain. The course of the bullet can be traced distinctly through these structures in the stereoscopic picture. I removed the bullet in a radical mastoid operation.

Plates showing a calcified tumor of the cerebellum, about three and one-half centimeters long and two centimeters thick. This case was under Dr. George W. Crile's care at Lakeside Hospital, and I saw it in consultation. The patient was in exceedingly bad condition when he was admitted to the hospital. Dr. Crile did a decompression operation, but the patient died later.

Plates showing temporosphenoidal abscess with gauze for drainage in the abscess cavity. The abscess was secondary to a chronic suppurative mastoiditis, and was located in the stereoscopic plates before the operation. I opened and drained the abscess through the mastoid wound. The plates which show the gauze in the abscess cavity were made one week after the operation. The patient recovered completely.

Plates showing necrosis over the sigmoid sinus secondary to an acute otitis media and mastoiditis. This case was promptly operated upon and recovered, although he had a beginning meningitis when he was admitted to the hospital.

Plates showing a tumor of the hypophysis extending into and nearly filling both sphenoidal cavities. This patient was nearly blind when he was admitted to the hospital, and refused any operative work except a decompression. This relieved his eye symptoms and headache.

The following description of the technic for making stereoroentgenograms of the head was given to me by Doctors Hill and Thomas, roentgenologists in the Medical Department of Western Reserve University.

TECHNIC FOR MAKING STEREOSCOPIC ANTEROPOSTERIOR RADIOGRAPHS THROUGH THE FRONTAL SINUSES.

The special apparatus needed for this purpose consists of a stereoscopic tube stand, a plate changer and an inclined plane the angle of elevation of which is about twenty-three and one-half degrees.

The plate changer is adjusted by means of the inclined plane so that it lies on the table at approximately twenty-three and one-half degrees elevation from the plane of the table. The tube is centered vertically, in reference to the table, over the middle point of this plate changer, and then adjustments are made so that when the tube is moved one and one-fourth inches to the right or left and the tube holder tilted, the central ray will strike the same point on the plate changer.

The patient is then placed on the table, face downward, with the forehead at the upper portion of the plate changer and the glabella over the central point. Care must be taken that the head lies squarely on the plate changer, so that the midsagittal plane through the head is at right angles to the plane of the plate changer.

The movement of the tube from one side of the head to the other is at right angles to this same midsagittal plane. The exposure made with the tube moved one and one-fourth inches to the right and the tube holder properly tilted, as above mentioned, will produce the right eye image, and the exposure made one and one-fourth inches to the left of the median line will produce the left eye image. The exposures are made during suspended respiration.

A compression cone diaphragm the size of the No. 1 Kelly-Koet diaphragm is used, and a heavy leather filter is placed over the patient's head.

TECHNIC FOR MAKING STEREOROENTGENOGRAMS OF BOTH MASTOIDS ON A SINGLE PAIR OF PLATES.

The patient lies on the table, on the left side, with the side of the head resting on a small table which is lead lined except over an area five inches wide across the center of the table. The ear is turned forward to prevent the shadow of the cartilages of the ear obscuring the roentgenogram of the mastoid. The head is placed so that the mastoid lies over a centering spot on the middle section of the table where there is no lead lining.

The tube is placed so that the line of the principal ray passes through the head at an angle of twenty degrees above a line through the auditory meati.

The stereoscopic adjustments have been made before the head is placed on the head table so the line of the principal ray shall always focus on the centering spot over which the mastoid is placed. Then on the left half of one eight by ten plate, marked "R" for instance, the right eye roentgenogram of the left mastoid is made. This plate is then removed from the slide in the head table without the patient's position being changed, the tube is shifted for the left eye image, and on the left half of a plate marked "L" is made the left eye roentgenogram of the left mastoid. The patient is then turned on the right side with the right mastoid placed over the centering point as was the left, and on the right half of the plate marked "L" the left eye roentgenogram of the right mastoid is made. This plate is then removed, the patient remains quiet, plate "R" is inserted in the slide of the head table so the exposure will come on the right half of the plate. The tube is shifted for the right eye, and a roentgenogram of the right mastoid for the right eye is made. This gives right eye roentgenograms of each mastoid on the plate marked "R," and the left eye roentgenograms of each mastoid on the plate marked "L."

These plates, when developed, are placed in the stereoscope with the one having the right eye images in the right light box of the stereoscope, and the one having the left eye images in the left light box. With the glass side of the plates turned toward the mirrors of the stereoscope we view the mastoids from the inside of the skull, while with the film side of the plates placed toward the mirrors we view the mastoids from the outside of the skull.

XL.

VOICE PRODUCTION FROM THE STANDPOINT OF THE LARYNGOLOGIST.*

By Hampton P. Howell, M. D.,

New York City.

Voice production is a rather hazy subject to the average laryngologist, unless he has devoted especial attention to it.

It would seem, therefore, that a short article on the principles of voice production and a consideration of its treatment relative to those who take up singing as a profession would prove of some value.

The writer's interest in this department began a dozen years ago, when he became an associate of one of the former presidents of this society, a man whose name is inseparably connected with the world of singers.

With the artistic side of the matter the laryngologist has nothing to do, but the physical effects of vocal methods do enter decidedly into his province.

Actual diseased or deformed structures throat specialists agree upon, but not so with functional conditions. By the laryngoscopic mirror alone we can no more pick out a noted tenor than we can an expert tennis player by examining his arm. In fact, by no means, does every singer of reputation have a pair of cords of normal pearly gray color. They may be somewhat dusky in hue, and the whole larynx may appear congested, and the thickened tissue in the posterior commissure be abundant enough to border on the pathologic.

In gross deformities and evident inflammatory diseases of the nose and throat, it is not difficult to decide on the best course of treatment. But in the complaint of a singer it requires very nice judgment to determine that a moderate hypertrophy of the lower or middle turbinal body, or that a slight thickening or deviation of the nasal septum, is the cause of the trouble. Nor can we say the disturbance is due to the shrunken remains of an adenoid.

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*Candidate's thesis to the American Laryngological, Rhinological and Otological Society, May, 1916.

Are you going to extirpate the slight enlargements of the lingual growth which you see?

Can you state positively that her small tonsils are diseased or interfere with the muscular action of the palatal curtain?

To the vocalist these decisions are of the utmost moment, for on the result of your sentence may rest an artist's future, especially if your management of the case demands operative means.

Considerable careful reading and practical experience, in the conditions common to singers and speakers, is necessary for any laryngologist who wishes to undertake this kind of work.

Of course, Tyndall's classic on "Sound," and von Meyer on the "Organs of Speech" are of fundamental value.

Of those authors who address themselves particularly to the departments in which we are interested, we would recommend especially the books of Sir Morell MacKenzie, H. Holbrook Curtis and Wesley Mills, dealing as they do with the mechanism and hygiene of the voice.

Too many of the treatises on these subjects have been written by singers and teachers, often of great renown, which appeal to the vocal student but do not handle the matter enough from the physiologic standpoint for a physician to follow.

The laryngologist should himself take lessons in singing and elocution and listen to instructions given to pupils. Much can be learned also by attending studio performances, the opera, and the study of the phonograph. Discussions, also, with those who are able to criticise the manifold problems and subtleties of voice method are highly profitable.

By such a course he trains his own vocal organs and his musical ear, so that he can not only test out a voice, but illustrate and give an exercise for a pupil to imitate with some degree of confidence. He will be able, also, to understand the sensations and feelings so often described by a teacher or singer to explain his ideas in connection with the production of tones.

Correct breathing is the basis of good singing; but the demands are quite different from those that are required for purposes of oxidation. We must take in air quickly and without noise, to meet the needs of the most exacting vocal phrasing. But more important than inspiration is proper expiration; how best to attain an even outflow of air under complete control, to permit just enough breath for the sound and no more, as demonstrated by the well known candle experiment —not a flicker of the flame held a few inches from the mouth during the emission of a sound.

Physiologic studies have demonstrated that the type of respiration so successfully used by the old Italian school is the one most preferred today. It earned its position among those natural teachers because it permitted the largest expansion of the chest with the best control of the air in expiration. This has been termed the "inferior costal type of breathing."

The socalled "upper chest," "diaphragmatic" and "abdominal" styles have all had their vogue at various periods. Each of these methods probably has a few supporters even now.

It is to be hoped that in future there will be a greater agreement on the underlying principles of voice production; that gradually there may be a closer cooperation between teacher and physician, all working towards a standardization in our methods of training voices.

To illustrate the "inferior costal type of breathing"—stand erect but not rigid, the shoulders gently thrown back, and somewhat raised, the upper chest immovable and the lower abdomen slightly contracted.

This position permits the best support in expiration, as the diaphragm is thus held tense and alert, allowing a continuous outflow of the air stream, with no unsteadiness or jerkiness. Having the upper chest, as it were, suspended and fixed, regardless of the posture of the singer, any demand made on the voice can be met.

While in everyday life we urge breathing through the nose, in the act of singing or making an address the case is different. Now we must breathe through the open mouth. Otherwise we could not take the air in rapidly enough. This method tends also to keep the tongue down in the floor of the mouth, with the necessary groove running back through its middle; and the laryngeal box is not pulled upwards, with a consequent tightening of the muscles of the neck.

The volume, or bigness, of a sound depends on the amplitude of its vibrations. A big bawling sound is most unpleasant to the ear, and it soon tires the auditory nerve.

Pitch depends on the number of vibrations in a given period. The healthy ear can distinguish sixteen vibrations per second as its lowest limit, and thirty-eight thousand for its highest. This is a stretch of eleven octaves. However, musical instruments have a range of seven octaves; and much of this is too high to cause a pleasing sensation on our centers of audition.

The average person can sing but two to two and one-half octaves, while in speaking we confine ourselves to a half octave of the middle voice.

The quality of a tone is affected largely by its overtones and harmonics. The blast of air through the glottis, in the case of the voice, sets the vocal bands in motion. This produces the primary sound. This simple neutral sound is somewhat enhanced by the ventricles of the larynx; it gains still more from the pharynx and buccal cavity; the low notes bring the chest walls into vibration as an additional resonator.

The peculiar quality or timbre of the individual voice arises from the accessory sinuses and the bony framework of the face.

Sometimes we are surprised to find in a robust man a small, squeaky voice. This is usually due to poorly developed facial cavities. The Maori of New Zealand possess peculiarly dead voices, though they have magnificent physiques. Anatomic researches show the lack of resonance in their voices is due to an underdevelopment of their accessory sinuses.

As a sound-producing apparatus the larynx is unique. Though comparable to the reed instrument, in the perfection and economy of its mechanism, it far surpasses it. This superiority is due to the fact that by the marvelous action of the laryngeal muscles a single modified reed can take the place of a great many, so placing in the confines of this small box a potentiality not to be found in a cathedral organ.

In the production of sound the larynx is steadied by its extrinsic muscles. During the utterance of deep notes, it is found to occupy a slightly lower position and rises in the ascending scale. The entire distance moved, however, is probably not over the fourth part of an inch. This rise and fall can be easily appreciated by the fingers on the thyroid cartilage during phonation.

Both in speaking and singing in the lower range of the voice, the cricothyroid is the chief muscle. Therefore we find it more used in men than in women, and in basses and baritones than in tenors.

By approximating the anterior part of the cricoid cartilage to the thyroid, it has the capacity of lengthening and making tense the vocal bands. This action can be noted when singing an ascending scale; for one can observe the arytenoid cartilages to move backward and almost out of sight, while there is at the same time a sinking of the posterior parts of the cords.

The thyroarytenoid muscle has a far more complicated action than that of merely drawing the arytenoid cartilage toward the thyroid. The inner fasciculus probably regulates the degree of tension and the elasticity of the vocal cords, possessing, also, the peculiar power of dampening the edges of the bands, just as a finger shortens the string of a violin in a high note; while the other fasciculus, on the other hand, narrows the rima glottidis—that is, it brings the borders closer together along their entire length.

The lateral and posterior cricoarytenoids, when acting alone, respectively approximate and separate the bands, but with the other laryngeal muscles they may operate as tensors.

When singing a high note the arytenoideus closes the posterior part of the glottic chink.

As the highest notes are reached there is also sphincter action about the entire superior orifice of the larynx, the whole organ appearing appreciably smaller.

If you ask a trained singer to run a scale from his low to his high notes, you will notice that when he reaches a certain point in his middle range a change begins from now on in the quality of his voice. It loses a certain loudness and open-ness and, as it were, sounds more covered up in the inside of the head.

This bridging over from the lower or chest notes to the head or covered notes may occur with a smoothness that makes the process unnoticeable, while on the other hand, the transition may be so pronounced that it causes a "break" in the voice. This alteration from a lower to a higher voice is designated as a change in the registers of the voice. Just as there is a change of gears in an automobile to accommodate itself to a change of speed, so there is a change of mechanism in the voice to adapt itself to the change in pitch.

According to Behnke, "a register is a series of tones which are produced by the same mechanism." A new register marks a change in the quality of a tone corresponding to the change in mechanism, and at the point where that variation occurs we term it the "break" in the voice.

In listening to a Sembrich there is such a perfection of method, gained through years of study, that an entire composition may be sung without the audience being aware of any such thing as registers.

Some teach that there are two registers, others three or even five. In fact, for every variety of note a different adjustment is probably required, though too slight to be perceptible.

But for practical purposes we can regard two registers as sufficient. We can add as a third the falsetto, which is not a genuine tone. This is a very high note of clear bird-like quality produced by male singers. It is created without the diaphragmatic support of the air column, and there is no dampening of the vocal bands in its formation; they are apart throughout their whole length, the vibration taking place on the very edges of the cords, if we are to believe Lehfeldt.

In the production of the falsetto there is a sudden readjustment from the previous high but natural note, followed by a sense of comfort in its performance. If well executed, or better, well disguised, it may prove of great utility on certain occasions.

To recapitulate: During phonation in the lower and middle range the vocal bands do not touch, as the cricothyroid is a tensor muscle, and the intrinsic muscles do not come into play during this part of the mechanism.

But as the scale ascends the cartilaginous glottis is closed by the arytenoideus, the cords come together more and more, both anteriorly and posteriorly, till on the very highest notes only a small oval opening is evident in the front third of the glottic fissure. It is at this period that the thyroarytenoid is effecting its complex adjustment, acting in conjunction with the lateral and posterior cricoarytenoids.

If the falsetto voice is used the cords again return to their original position; that is, apart for their entire length.

As previously stated, to give one an understanding of the application of registers, without some practice under a teacher, is most difficult.

There are certain terms used in the singing world of which we as laryngologists must gain a working knowledge. The covered voice was described with the discussion of registers. We should appreciate the difference between singing "too white" and "dans le masque." In the former method the voice is brought too far forward in the mouth; the tones are clear cut but colorless, and they do not charm, for they lack overtones. In the tones sung too "dans le masque" the voice is confined in the facial cavities, and where we have this to excess the quality of the tones becomes too covered and "hooty." This shuts up the voice, prevents singing high notes and interferes with clear diction. This is not a difficult fault to rectify, but sufficient to keep a good voice in the ranks of mediocrity, if continued.

The term "stroke of the glottis," or "coup de glotte," originally referred to the correct attack of a tone, the proper adjustment of the tension of the vocal cords to the column of expired air. Its meaning now is quite the opposite: a sudden attack with the throat muscles rigid and the vocal cords overtense, with margins that touch along their middle in the production of the sound. This is a serious fault, and one that is certain to bring disaster to the voice.

The tremulo in good singing today is never justified, and ordinarily we find it is a defect due to an uncertain attack and a poor respiratory support.

Far more than the actor, a singer must hold his emotions under control. The gallery loves a throbbing open tone on a top note, but the man who commits this injury to himself will soon require the throat specialist.

At what age should vocal lessons begin? We may answer: At any time; the earlier the better, but with strict limitations in the compass of the voice, as at a tender age the organ cannot withstand the strain of unusual effort.

Still we must bear in mind that in early life the memory and initiative faculties are at their best, and these natural characteristics should be taken advantage of.

Choral singing, in our institutions, on the other hand, is harmful. For, though it may teach the elements of music, no attention is given to tone placement; the pupil sings beyond his range, and each one tries to outdo his neighbor. This procedure often results in voice strain and blasts forever an innately excellent organ.

Were an attention, at all proportional to its importance, given to expressive reading and elocution in our schools, we would not find so many adults with wooden voices and slovenly methods of speech. What greater possession than a well modulated voice and clear enunciation. And yet how few appreciate these facts?

We must employ great judgment in advising the use of the voice during the early period of adolescence, when the larynx is undergoing its most rapid growth. If the alteration is gradual, moderate exercises may be permitted, but within prescribed limits. Voices, however, which are unsteady or husky at this age, which often "crack," show evidences of laryngeal congestion, and in such cases singing or any harsh use of the vocal apparatus must be interdicted for a year or more, if necessary.

Really not until the age of twenty-three or four does the larynx reach its full development, when it is competent to endure serious vocal training.

Every laryngologist should know several reliable vocal teachers, to whom he can intrust his musical patients.

In the fundamentals of voice production, we can state that a man is the better teacher for a man, and a woman for a woman.

Be most particular in the selection of your first teacher, for a pupil should be so trained from the beginning that he will not be compelled to unlearn incorrect methods later on. Studios are filled with those who have made such a mistake.

Ordinarily one can judge best of a teacher by the recognized results he has had with his pupils.

Do they sing well? Do their voices improve? These are the first questions to ask.

Remember, we laryngologists are not singing teachers. With the divine art of song we have nothing to do in the consulting room. But whatever pertains to the actual voice, the effects from good or bad instruction, as noted on the vocal apparatus, do decidedly fall within our jurisdiction.

To excel as a teacher he must know his subject, have unbounded enthusiasm, and possess a sympathetic insight into the needs of his pupils.

It is unnecessary for him to be an eminent singer. In fact, the drudgery of teaching tone work would very often be repellent to anyone fitted for the concert, stage, or opera.

Still he must have a voice so trained that he can demonstrate a correct tone, and it is a great advantage to imitate a student's own mistakes and then to sing the part faultlessly to him. It is often far superior to a long explanation and better keeps up the interest of the pupil.

The best teacher depends on his ear, and not on the laryngoscope. He should not regard himself as an instructor of anatomy and physiology. To indicate any more than the gross divisions of anatomic science to a pupil will not assist him; rather, it will make him overconscious than otherwise.

To bully and tyrannize over his pupils is absolutely wrong; for a singer's nature is altogether too sensitive to endure such rough measures. The nervous organization of one of our great singers is of different mould from that of the humdrum individual. We average mortals try to explain that difference by the words "artistic temperament." At any rate, we know an artist possesses a spark that reaches out into spaces quite unknown to most of us.

If a pupil fails to make progress, if he is hoarse after his exercises, has difficulty in understanding his teacher, and is becoming discouraged, you can be certain that he must find his instruction with someone else.

Let us now consider a few types of cases such as a throat man might have visit his office.

Case 1 is that of a young man, a tenor in a church choir.

He states that his first teacher considered him a baritone who had some tenor notes. With this man he made excellent progress, and his exercises and songs were kept well within his range. Two years ago he met a teacher who classed him as a robust tenor. This prospect seemed too alluring to neglect, so he put himself under his direction. For a time he improved, at least he said his range was higher by several notes; and though he had not yet reached high C, he hoped soon to do so.

His first serious trouble began when he received a church position. The music always seemed too high for him, though by an effort he got through his parts. Of late, however, he noticed a tremulo in his voice, and a certain unsteadiness, and often after a performance his throat ached, and there was a huskiness in speaking. He also added that he is now losing his lower notes, and that the general quality of his whole voice is not so good.

You ask the young man try a scale and you find his higher notes lose their freedom; they sound pinched. Having him run down the measure, you recognize at once that here lies his natural voice, though it sounds wooden.

On examining his nose, you find a high deviation of the septum, while the cords are somewhat wavy in outline—and that in singing a high note on "Day" or "Hay" you see the glottis is open its entire length.

Such an examination clearly proves that this patient is not a tenor; that he has been forcing his voice so that the inner fibers of his thyroarytenoid no longer act. The "long reed" type of glottis still further confirms the idea that the patient has a voice of medium range only.

The high septal deformity does perhaps interfere with his highest notes, but as those do not belong to his natural range, no operation on the septum is necessary, especially as the septum is producing no other symptoms.

The conclusions to be drawn are evident. The man has attempted to outdo nature. You cannot create a voice; you can only improve the one you already possess.

Let his voice drop back to the place where it properly belongs, and again he will sing as he did formerly, as thus far no permanent injury has been done.

Case 2 is that of a young comic opera star, whose company opened two weeks ago. After singing for a week she thought she had contracted a slight cold. Her middle voice and piano notes are poor, while her high notes are as good as ever. She is hoarse after a performance.

Her nose and pharynx manifest slight evidences of congestion. The larynx is in the same condition; the cords have a dusky hue and bulge towards the center. On light phonation one observes a single spot just anterior to the middle where the mucus adheres. Under this on the margin of each cord is a minute tumefaction with an area of redness about it. Closer study of these growths shows them to be the size of a pin-head; they appear harder than the surrounding tissue, and seem embedded in it. Their location is exactly at the point where the bulging cords touch.

The diagnosis is a traumatic laryngitis with singer's nodes, due to faulty voice production.

The supposed cold is merely the congested state of the upper respiratory tract resulting from the voice strain.

In such cases the upper notes remain clear, because when the cords are tense the edges do not rub. But in the middle voice and piano notes, the cords are relaxed and at their most convex point, from the attrition of the edges, the nodules quickly develop.

In all cases of vocal disability due to voice strain, examine for the bulging margins of the bands.

The little vibrating pearl of mucus reveals the point of origin of the vocal nodule.

Light opera music is not written with the thought of saving voices, but rather for striking vocal effects.

Is it any wonder that the prima donna, often selected for her beauty and youth, breaks down early in her career? It takes six years of painstaking work to prepare a singer for the operatic stage—and yet for a light opera the principal star is frequently selected before she has grasped the rudiments of voice culture.

What are the indications for treatment in a case of this kind? Local measures and prolonged rest, you may say.

Applications, inhalations and sprays have little effect, except to soothe or act as slight counter irritants. Prolonged rest is out of the question with this class of people. They must sing or they lose the place they have so labored to attain.

The exercises instituted and developed by Dr. H. Holbrook Curtis bring the most satisfactory results in the shortest time. Numerous articles of his have thoroughly explained this method of treatment in the voice strain of singers.

The general idea is that instead of giving the vocal organs complete rest, the patients are to be given exercises to counteract the damage done—in the same fashion as one who has tired his arms by climbing a rope can quickly restore their elasticity and vigor by using the punch bag. For wrong methods of singing, which brought the cords together, are substituted those that exercise and in a way massage them, but do not permit them to touch.

The patient is requested to whisper instead of being allowed to talk. He is to practice humming exercises every hour during the day for a few minutes.

To briefly describe the technic of performing the hum: We have the patient sip in some air through the mouth, then close the lips, with the teeth a pencil's breadth apart, think of the word "maw" or "naugh," in the most comfortable pitch of his middle voice, and then first sing the word in the closed mouth, bringing it forward till you can feel the lips vibrate with the sound, when by flecking the under lip the explosive note "maw" or "naugh" escapes.

By ascertaining in this manner that the sound is correctly placed, let it, as it were, burst through the lips. It should be clear and even. After some practice he can go up and down the scale with it, and then in a day or two he may try his songs, using, however, the word "maw" at first in place of the words of the song.

In a remarkably brief period voices have been restored by this method, and the patients been enabled to return to their occupations.

Case 3 has many of the symptoms of the former patient: the voice tires easily; it is losing its attractive quality, and there seems a certain breathiness of tone.

By laryngoscopic examination you find cords that are neither wavy in outline nor bulging. Instead, they are bowed, with an elliptical opening.

Such a condition arises from voice strain also; too much air is used in the production of a tone, till finally the thyroarytenoid muscle, yielding to the strain, causes the oval opening of the glottis with a weak, breathy result. It can be occasioned, too, by practicing exercises excessively that contain too many open vowels.

The principles of treatment here are to properly apportion the amount of air to the needs of the voice, and to make use of the exercises suggested for the benefit of the previous case.

It is to be hoped by the writer that even though every laryngologist is not endowed with a keen musical taste, he need not consider that the subject belongs to a domain in which he can have no part; that he will discover, by applying his anatomic and physiologic knowledge to the principles of voice production, and by training his own ear and voice, he may find this branch of his specialty of increasing interest and profit to himself as well as to the vocalist.

XLI.

INTRANASAL DRAINAGE OF THE FRONTAL SINUS.

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History: The first references to intranasal drainage of the frontal sinus that I can find are in Jonathan Wright's "History of Laryngology and Rhinology," from which I quote: "Ogston in 1884 declared that there was no possibility of introducing a probe into the frontal sinus from the naris; but subsequent work slowly demonstrated the error of this dictum."

Schaefer in 1890 proposed to drain the frontal sinus by enlarging the nasofrontal duct. He was followed by Winckler in 1892, and in 1893 Lichtwitz again urged the practicability of this operation, but for years the idea was discredited by most writers as dangerous or even impossible; but Winckler's work in 1892 did much to show the practicability of the operation. However, on account of the difficulties he experienced in passing a probe through the nasofrontal duct, he recommended piercing the floor of the sinus between the anterior end of the middle turbinate body and the septum, for which he used a curved sound with a dull point.

In 1898 Scheier reiterated that it was practicable to enter the frontal sinus through the nasofrontal duct.

Spiess also advocated the procedure in 1899. However, none of these operations found favor with the profession, and, as stated by Wright, "when an operation was necessary, the whole drift of opinion for ten years was toward gaining access to the sinus by means of an external operation."

After giving due credit to many workers for the development of the external operation, Wright refers to the symposium at the meeting of the American Laryngological Assocaition in 1905 on diseases of the sinus and the operations for its alleviation. He mentions my report at that time, and subsequent reports by myself and Skillern in 1907 and 1908.

In 1906 Halle of Germany reported operations on ten persons, for enlarging the nasofrontal duct by an electrically driven burr, in which operation he protected the internal and posterior walls of the cavity by a flattened director, held internally and behind the burr. In this paper he refers to the methods of Schäffer, Hajek and Grünwald, by introducing metal sounds into the nasofrontal duct after removing the anterior third of the middle turbinated body; but he considered the operation difficult and often impossible. However, none of these really enlarged the drainage canal. In this same paper he referred to my operation as the only one that completely opened the nasofrontal duct; but he thought it dangerous on account of coming too near the tabula interna, a fear which my experience has demonstrated to be unfounded.

In 1908 R. H. Good of Chicago, and Vacher in France in 1910, advocated intranasal drainage. Referring to the internal operations advocated by these various writers, Wright says "they met with considerable criticism; but there is no doubt that in selected cases it has become the operation of choice with many rhinologists." In 1910 J. A. Thompson of Cincinnati proposed enlarging the nasofrontal duct by means of bent rasps. Good used rasps, chisels and biting forceps to cut away the nasal crest. Thompson, in his operation, designed to have the mucous membrane on the posterior aspect of the canal left intact, to promote quicker covering of the enlarged canal with mucous membrane. He also used a director and had a groove on the back of his pointed rasp which he works along this director so that he may enter a smaller nasofrontal duct than could be done with the Good rasps. Segura in 1912 and Sullivan and Denis the same year advocated changes in the operations which they considered useful advances.

In 1912 H. P. Mosher, at the American Laryngological Association, recommended breaking through the anterior end of the middle turbinated body with a curette and working outward, backward and downward, breaking down the cells and the nasofrontal duct which is surrounded by them, destroying both the cells and the duct at the same time. He says: "Very little curetting is required to convert the anterior part of the labyrinth into a single cavity. In the roof of this chamber, usually in the anterior outer angle, the opening of the nasofrontal duct is placed."

In 1914, in the *Journal of Laryngology, Rhinology and Otology* for May, P. Watson-Williams, in an excellent article, presents a good historical sketch of the intranasal operation, with fairly accurate descriptions of the various operations, and describes his modifications of the other procedures. In the same number Herbert Tilley described his method, and refers with praise to Mosher's operation. He takes the position that when any operation is necessary an efficient intranasal operation is to be preferred unless symptoms indicate intracranial complications, unless necrosis or osteomyelitis are present, or unless ocular complications render a radical operation necessary; but he admits that anatomic conditions may interfere with the intranasal operation, and he calls attention to the fact, now commonly recognized, that not a few cases of frontal sinuitis require no operation at all, and that others recover promptly after removal of the anterior end of the middle turbinated body.

In 1915 Dr. O. T. Freer presented a paper to the American Laryngological Association, on opening the frontal sinus through the nose. He gives a careful exposition of the anatomy, and points out the dangers both of rasps and burr, but states that the latter properly used is a safe instrument. He thinks the drainage canal usually obtained is too small and becomes constricted by cicatricial bands. However, this cannot occur if the gold drainage tube is left in position, as I recommend. He favors enlarging the ostium backward through the foveal ethmoid cells, which may be easily broken down. He recommends numerous probes and small curettes which he uses in cleaning out the ethmoids and frontal sinus. He states that there is very little danger of perforating the cranial covering of the ethmoid cells, which is hard, though thin, unless it is directly attacked. Externally, however, the orbital wall is frail and is liable to perforation in any of the intranasal operations.

Without disparaging any of these operations, they do not appear to me to make adequate provision for keeping the canal open, excepting by its large size, and as we know from experience with other accessory nasal sinuses there is a strong predilection for closure of artificial drainage openings, even though these may have been large.

Many laryngologists have considered it necessary to curette the frontal sinus in nearly all cases to remove hypertrophy, polypi, degenerated mucous membrane, exuberant granulations, and what Herbert Tilley calls the pyogenic membrane, but I am pleased to find that Freer agrees with the opinion I have held since the early days of intranasal drainage. He says: "At first I was also under this impression, but experience has shown me that not only are these advanced degenerations exceptional, but that the mucous lining of these sinuses is usually normal in appearance, and that even if the pathologic changes mentioned do exist, they are capable of disappearance under the influence of good drainage and ventilation unless there be caries of the bone or large polypi present." He does not, therefore, favor systematic destruction of the mucous membrane, which he thinks would prolong the period of recovery and suppuration. I heartily agree with this view, and from experience believe that in a large majority of cases free drainage is all that is necessary, and that vigorous curettage is a bad practice, although gently removing polypi or granulation tissue with the curette, when these are present, would undoubtedly hasten the cure.

In 1905 I gave a careful description of my operation, and it has been described by several others in journals and textbooks with varying degrees of inaccuracy. I would not write about it again had it not been for a grotesquely inaccurate report of some remarks I made on the subject that appeared in an abstract of our proceedings of two years ago. This report muddled the whole matter beyond recognition by anyone not fully familiar with the operation. But there are also some new points suggested by my experience that are deserving of notice.

I hope now to make the matter so clear that the operation can be readily and safely done by anyone qualified to practice laryngology and rhinology. The furor for extensive mutilating operations on the frontal sinus has had its day, reached its zenith, and declined until now a large number of laryngorhinologists favor more conservative intranasal drainage in suitable cases, and have come back to the well known principle held by our forefathers, viz., that a suppurating cavity properly drained will usually heal if meddlesome surgery is avoided.

In 1893 I had under my care two cases of chronic long standing suppuration of the frontal sinus. Both of these patients lived out of the city, and they came to me irregularly at intervals of weeks or several months. I gave them such relief as possible by detergent washes and occasional washing out of the frontal sinus, but neither of them would consent to an external operation. Finally, in one of these cases, by repeated washing out of the sinus daily for a short time, and after a couple of weeks less frequently, and by following this washing with a five per cent solution of protargol injected into the sinus, I succeeded in effecting a cure, although the disease had persisted for twelve years. There was no recurrence of the empyema in this case.

In the other case it was not so easy to treat the sinus locally, and for a long time I pondered upon some effective method for enlarging the nasofrontal duct.

In 1895 the late Dr. A. W. Palmer of New York devised a frontal sinus drill which was described in the *New York Medical Journal,* March 2, 1895. This drill was illustrated in the third and fourth editions of Dr. Kyle's book, and in the third and fourth editions of my own work. It possessed some elements of success, and at first sight appeared practical; but it had no safety guide to confine it to the nasofrontal duct, and I do not know that it was ever used. At any rate, it did not obtain popular recognition. I think, however, that I must be in a way indebted to Dr. Palmer for the idea which finally evolved into the burr which I invented. I concluded that if a perforated, bead-like burr could be devised to be run into the nasofrontal duct over a small director, or safety guide, this duct could be enlarged so as to furnish adequate drainage. After several experiments I succeeded in developing such a burr that filled the requirements and that has needed no material alterations. I tried this out on the cadaver, and had it altered as needed until I was finally satisfied. This burr made a canal six millimeters in diameter; and the guide was so arranged as to prevent any danger of the burr passing through the posterior wall of the frontal sinus. But I soon learned that a canal of this size left to nature would not remain open more than a few weeks, therefore, I fashioned a metal tube that I inserted into the canal, which I hoped to keep in place by bending out sections of both its upper and lower ends.

On March 25, 1904, I operated on the first living subject by this new method. This was the second of the two who had so persistently declined external operative measures. The drill worked perfectly and a good drainage canal was quickly formed, but I had much more difficulty than I had expected in placing the drainage tube; indeed, it was a failure, and I had to remove it after a couple of days. The canal in this case had so far closed at the end of five or six weeks that all of the old symptoms returned, and I found that only a very small director could be introduced.

In the meantime I had perfected the spring-gold self-retaining tube that I still use. Introducing the small steel guide, I ran in the burr again and then inserted the gold tube, which gave perfect drainage and allowed the patient herself to easily wash out the frontal sinus. As a result the sinus completely healed in a few months, although it had been suppurating for sixteen years. The patient was so timid and so feared a return of her old headaches that she would not permit me to remove the tube until she had worn it three and one-half years, although it had not been needed for more than about three months, for in that time the drainage canal had been completely lined by mucous membrane and there was no possibility of its closure.

As has been aptly stated by someone whose name I cannot recall, the efficiency of an operation is not determined by what can be done with it by its author, but by what can be accomplished with it in the hands of other surgeons. I am convinced that my operation can readily be performed by any properly equipped laryngologist, and that it will prove effective in nearly all cases of chronic suppuration of the frontal sinus; indeed, I have placed the percentage as high as ninety-five per cent of suitable cases, and I have no reason to modify this statement; but of course in a pansinuitis involving the ethmoids and sphenoidal sinuses it cannot be sufficient. In certain cases of polypi or other growths in the frontal sinus it will not be sufficient, but it will establish permanent drainage even in these, and with such drainage many of them will recover without further interference.

The essential instruments for my operation are fine steel bent probes, 14.3 centimeters in length by .6 millimeters to 1.5 millimeters in diameter (H to H[1], Figure 1), that are introduced, with the aid of a removable handle, through the nasofrontal duct into the frontal sinus; a perforated or (beadlike) burr (G, Figure 1), that is run in over this probe to make a drainage canal of adequate size, and a spring-gold self-retaining tube (E, Figure 1), to maintain the lumen of this canal until it has become covered with mucous membrane.

A similar tube as large as the canal made by the operation would, I think, be a great help in the subsequent treatment in all of the other practicable intranasal operations. This tube not only prevents closure of the canal, but it is a great help to the patient in washing out the frontal sinus, which can be done every day after the operation by the patient himself. With the eustachian tube catheter that I give the patient for washing out the sinus he can readily locate the metal drainage tube and irrigate the sinus without pain. In order that this operation may be made perfectly clear, I must repeat something of what I wrote a decade ago.

I first attach a silver canula 1.5 millimeters in diameter to a two dram syringe (B, Figure 1) and pass it as far as possible into the nasofrontal duct, the lower orifice of which is immediately behind the uncinate process and external to the anterior end of the middle turbinated body. This canula is ten centimeters long, and in order to allow it to adapt itself to the sometimes tortuous frontal sinus, three centimeters of its distal end is of soft silver. In order to avoid pain and to remove swelling the syringe is charged with ten per cent of cocain in 1/4,000 of epinephrin, and as the canula is introduced about one minim of this solution is forced through it, about every two minutes, three or four times. In the great majority of cases the canula may after a few minutes be carried on into the frontal sinus, but sometimes the enlarged agger cells that occupy the upper part of the uncinate process obstruct the passage, and in other cases the canula passes into a frontoethmoid cell and it is not possible to follow the nasofrontal canal; however, the region has thus been anesthetized and the swelling reduced. Then I try the steel probes and alter the curve as seems indicated by the direction the silver canula has taken. If in the adult the end of this probe cannot be passed more than 5.2 centimeters from the posterior inner angle of the nostril, I know that it has not gone into the frontal sinus. When it passes into the sinus, the end of a probe bent from thirty to fifty degrees will be six or seven centimeters from the point indicated.

In order to avoid all danger, frontal and lateral skiagrams should be taken with the probe in situ, and thus one may be certain whether or not it is safe to enlarge the canal to a diameter of six millimeters. (Figures 2 and 3.) I have examined a great number of anatomic specimens with this point in view, and have seen only one where the burr could not have been passed with perfect safety. In that one the nasofrontal duct ran so close to the olfactory groove that almost any operation, either internal or external, to establish drainage would have opened into this groove.

In all cases the anterior third of the middle turbinated body should be removed before the operation. This allows more freedom of motion. I have done the operation two or three times without this procedure, but have regretted it, as swelling of this tissue is apt to interfere with free drainage and with irrigation. If possible the frontal sinus should be well washed out before the operation is done. After the safety of the operation has been proven by the skiagram, with the probe in position, the nasofrontal duct and the frontal sinus are anesthetized by injecting into the sinus about a dram of five per cent cocain in 1/4,000 of epinephrin. The whole naris is then thoroughly swabbed with vaselin. The probe, which should be of a length to project four or five millimeters beyond the burr (G, Figure 1) when the latter has been passed over it to the farthest extent, is then introduced and its handle removed. The shank of the burr is then attached to the chuck of the dental engine, and the perforated burr with the spiral metal tube (F, Figure 1) over its shank is slipped over the probe and pressed up to the roof of the naris. The current is then turned on, slight pressure is made, and in a second or two the frontal sinus has been opened.* I usually pass the burr up and down a couple of times to clear the opening of any obstructing mucous membrane, and then the operation is finished excepting what might be termed the dressing.

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*A simple handle may be used instead of the dental engine, and the burr run in by hand, which would require only a little more time in most cases.

In any of these intranasal operations it is very important to caution the patient not to blow the nose, because of the danger of forcing air and septic material into the orbit or subcutaneous cellular tissue.

In this operation there is very little blood, so that the field of operation is not obscured. When there appears to be any danger of injuring the posterior wall of the frontal sinus, a strong thread is fastened to the distal end of the spiral tube (F) and kept from slipping by a narrow strip of adhesive plaster, and when the burr has been passed up to the roof of the naris, strong traction is made on this thread so as to draw it and the burr forward. When this is done, or when the probe has had to be bent more than usual, the enlarged canal has a curve that may not admit the other instruments or the gold tube, therefore, in such cases, as soon as the burr has been withdrawn, I introduce another probe with the normal curve (H^1, Figure 1), and then run the burr in again. I devised a shield to use in such cases, but now think this simple method better.

In cases where the end of the probe has been caught in a frontoethmoid cell or stopped by the upper end of the processus uncinatus, I pass it in as far as practicable and then run in the burr, after which I have never yet found any difficulty in passing the probe into the frontal sinus and completing the operation.

When the burr has been removed I pass in the bent tube packer (D, Figure 1), through which I introduce the end of a yard of a half-inch strip of gauze, about a foot of which has been saturated with a liquid formed by rubbing together equal parts of camphor and carbolic acid. This is a powerful antiseptic but not an escharotic. It is pushed on into the frontal sinus and allowed to remain about five minutes. The packer tube is then drawn down about half an inch so as to expose the raw surface caused by the burr, and the gauze is slowly withdrawn, thus practically searing the raw surface so as to prevent absorption of the pus that will for some time flow down from the frontal sinus, (Figures 4 and 5.)

CASE RECORD.

McF., A. O.—Figures 2, 3, 4 and 5 show the skiagraphs of an unusually difficult case of two months' duration, following a crushing injury of the nose, forehead and chest that had occurred ten months previously. The wounds had mostly healed, leaving great deformity which dislocated the landmarks so that it was difficult to find the orifice of the nasofrontal duct, and which had so changed the direction of the latter as to require an unusually sharp curve in the canula, as shown in Figure 3. This should be compared with the normal curve H^1, Figure 1.

In this case the end of the silver canula was engaged in the opening of the nasofrontal duct and a couple of minims of a ten per cent solution of cocain in 1 to 8,000 of epinephrin were injected three or four times at intervals of about two minutes, which reduced the swelling so that the canula was finally forced into the frontal sinus, as shown in Figure 3. This canula was only one and one-half millimeters in diameter. Circumstances made it necessary to delay the operation two days, and then the nasofrontal duct was so much more swollen that the canula could not be inserted more than eight millimeters, which appeared to be about one-third of the length of the canal.

After as good anesthesia as practicable was secured the steel probe was inserted to the same depth and the burr run in, after which the probe could be inserted into the frontal sinus and the burr was run in the whole distance; but the curve of the canal thus made was so sharp that it had to be reduced by rasping out the anterior portion of the canal with Thompson's instrument. The gold tube was then introduced, but, as shown in Figure 4, it was too long, and the drainage canal was so large that it was not securely held in place. In this instance it was difficult to see the anterior end of the middle turbinated body, which was the only landmark for finding the nasofrontal duct, therefore this was not removed until after the burr had been passed into the frontal sinus.

To prevent bleeding from this latter wound, and at the same time prevent the gold tube from slipping down, the naris was packed with a strip of rubber sponge two and one-half centimeters wide, six centimeters long, and five or six millimeters thick. This packing is shown by the peculiar cellular appearance in the naris below the tube in Figure 4.

Some days later, when the swelling about the wound to the turbinated body had gone down, the original gold drainage tube was withdrawn and a shorter one inserted (Figure 5).

In cases where I have reason to suspect polypi, granulations or swollen degenerated mucous membrane, a small flexible curette (A, Figure 1) is used carefully before applying the gauze. The vaselin that has been swabbed over the naris protects the mucous membrane from any irritating effects of the antiseptic. As the gauze is used in a single strip, there is no danger of leaving any part of it in the frontal sinus unless it should be torn; however, it would be well to measure it before and afterward.

The spring ends of the gold tube (E, Figure 1) are pressed together by winding an elastic band around them, and slipped into a No. 2 gelatin capsule. The spiral tube (F) is then placed over the applicator (C, Figure 1), and the gold tube is slipped on its distal end. Thus the gold tube is easily introduced, and the spiral tube is pushed up to hold it while the applicator is withdrawn. I then introduce the small steel probe once more, to punch a hole in or push off the gelatin capsule, which will dissolve in three or four minutes. The gold springs at once spread out and hold the drainage tube in position. If one has thought best to enlarge the opening beyond the size of the burr, on account of diseased surrounding cells, the gold tube should be of sufficient size to fill the canal, otherwise it may not remain in place. Latterly, I have dipped the gelatin capsule, after being applied over the gold tube, in paraffin with melting point of 105 to 115 degrees F. before introduction, so that it will not dissolve before I have the drainage tube in position. This paraffin is broken by the expansion of the gold springs and very soon escapes with the secretions. The operation is thus completed. The patient has had the minimum of shock and pain; there has been no loss of blood; all the steps of the operation have been carried out under accurate observation, and the patient is not required to remain in the hospital, for he can wash out the frontal sinus as often as desired, without pain. For this purpose I give the patient a metal eustachian catheter, bent to the same form as the applicator, which I mark by placing about it a strip of rubber plaster that will come to the nostril at its upper part when the end of the tube is at the orifice of the drainage tube; then the contact of metal on metal will be recognized and the end of the catheter can be easily slipped into the drainage tube. A hand bulb with fixture to fit the flaring end of the eustachian catheter is used to wash out the cavity. The mucous membrane from the frontal sinus, from the naris, and from the islands of mucous membrane opened up in surrounding cells, soon spreads out outside the gold tube and forms a complete mucous covering, thus leaving a canal six millimeters in diameter that cannot close and that approaches as nearly as possible to normal conditions. This is an important factor in preventing reinfection of the sinus when the patient subsequently gets colds in the head.

The drainage tube should be left in three or four months. It gives the patient no inconvenience. This operation in no way militates against a later external operation, if that should be thought necessary; indeed, it is an aid to the external operation, as a large drainage canal has already been made into the naris, and it in no way interferes with subsequent operations on the ethmoid cells.

No operation in this region is free from danger, and the greater the trauma the greater the risk. From my experience I believe this operation is the safest, and that it will prove efficient in nearly all chronic cases, and that patients will recover as quickly as from any of the other operations.

In acute cases it is usually much more difficult to pass the probe, and when this cannot be done the operation is not applicable until some of the anterior ethmoid or the agger cells have been broken down by Mosher's or Watson-Williams procedures.

It should be remembered that the nasal ostium of the drainage canal from the frontal sinus is located just external to the anterior end of the middle turbinated body, and that not infrequently, in both acute and chronic inflammations of the frontal sinus, removal of a part of this turbinal will relieve the obstruction and insure recovery.

In a large percentage of acute cases the application of heat externally, and especially by the Bruenings headlight bath (Figure 6), frequent applications of appropriate quantities of epinephrin and cocain will reduce the swelling so as to avoid the necessity of any operation.

In acute inflammation of the accessory sinuses we obtain much benefit by the repeated use of heat, which is most conveniently applied by the headlight bath. The patient should be kept in bed and the bath applied from once to four times a day, according to the severity of the symptoms.

The electric bulbs are used to furnish heat, but probably have no other influence.

In using the light bath the eyes are thoroughly protected from the light. The box is placed over the head and the current is turned on and left until the thermometer shows 182 degrees F., which usually requires about fifteen minutes. The current is then turned off, but the bath is continued about fifteen minutes longer until the temperature comes down to that of the surrounding atmosphere.

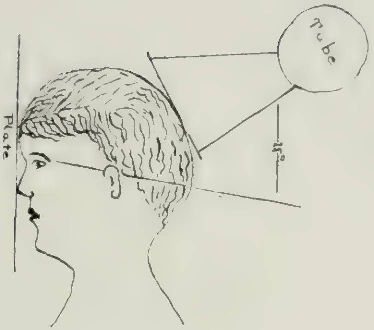

FIGURE 1.

This shows the position of the patient's head on the plate and the angle at which the tube is placed for taking stereoroentgenograms of the nasal accessory sinuses.

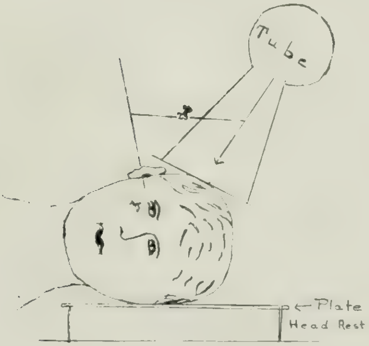

FIGURE 2.

This shows the position of the patient's head and the angle at which the tube is placed for taking stereoroentgenograms of the mastoid.

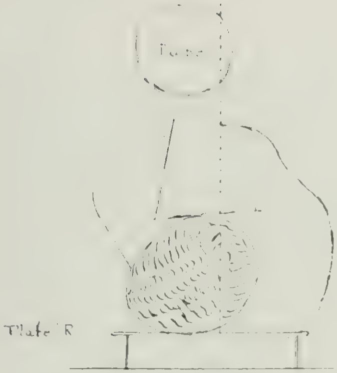

FIGURE 3.

Position No. 1. Right stereo of the left mastoid.

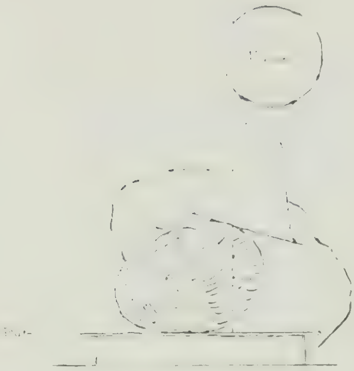

FIGURE 4.

Position No. 2. Left stereo of the left mastoid.

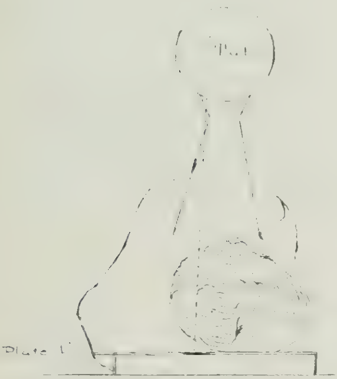

FIGURE 5.

Position No. 3. Left stereo of the right mastoid.

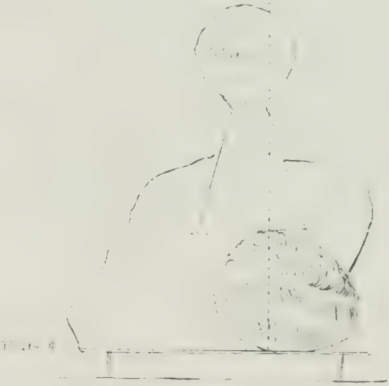

FIGURE 6.

Position No. 4. Right stereo of the right mastoid.

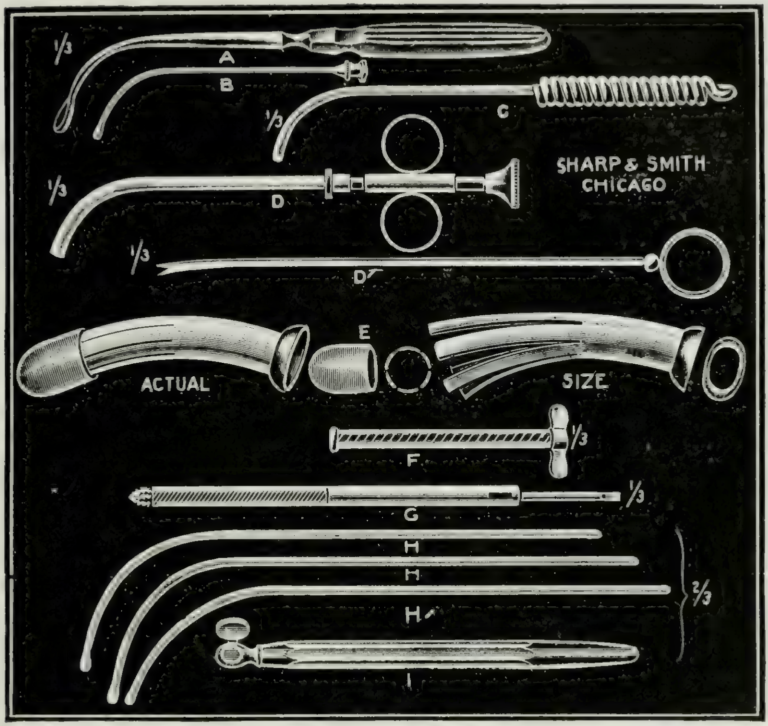

FIGURE 1.

A, curette; B, silver canula for syringe; C, applicator; D and D¹, packer; E, gold drainage tube—from left to right, first with spring ends covered by gelatine capsule, capsule, lumen of tube when springs are closed, tube with springs expanded and end of tube; F, spiral tube; G, perforated or bead-like burr; H, H, steel directors or probes, of different sizes and bent at different curves; H¹, steel director of normal curve; I, handle for directors or probes.

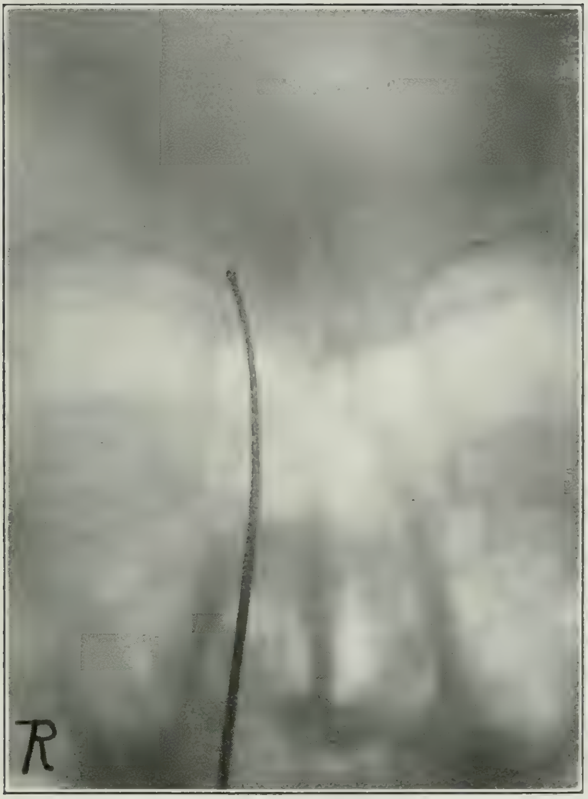

FIGURE 2.

Radiograph of case here recorded with canula in right frontal sinus. Outlines of right sinus obscured by pus. Left sinus small.

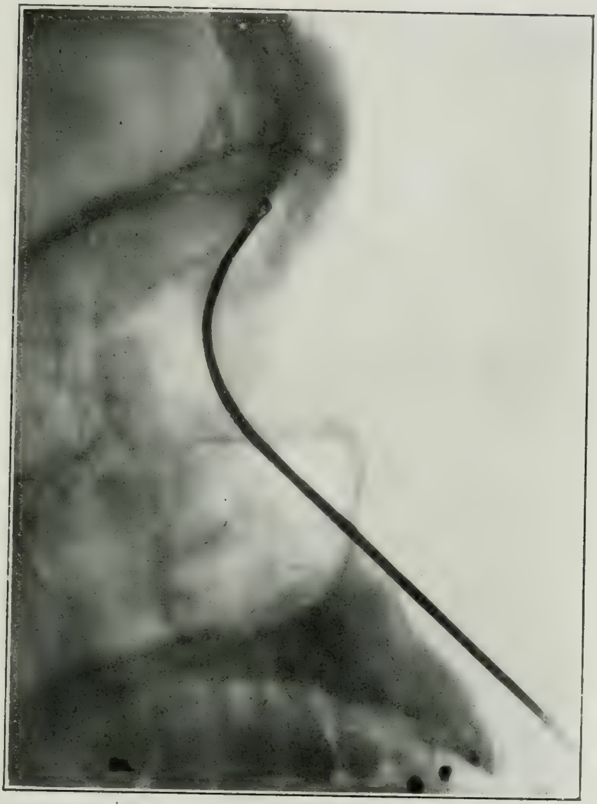

FIGURE 3.

Lateral view, canula in right frontal sinus. These two views show that the burr can be introduced without danger. Note extreme curve of the canula, as compared with normal curve H^1, Figure 1.

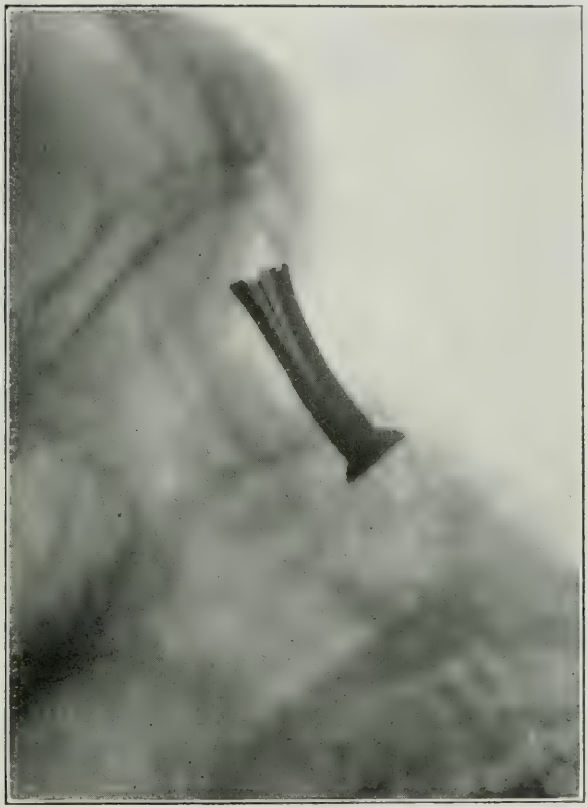

FIGURE 4.

Same case shortly after gold drainage tube had been introduced. Note that the tube was too long. The cellular appearance below tube is due to packing of rubber sponge.

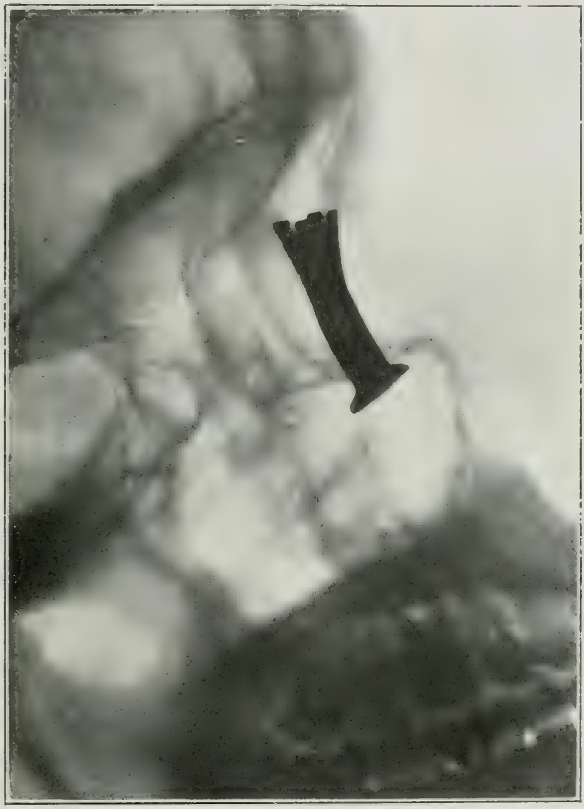

FIGURE 5.

Same case with shorter gold tube, which is in correct position.

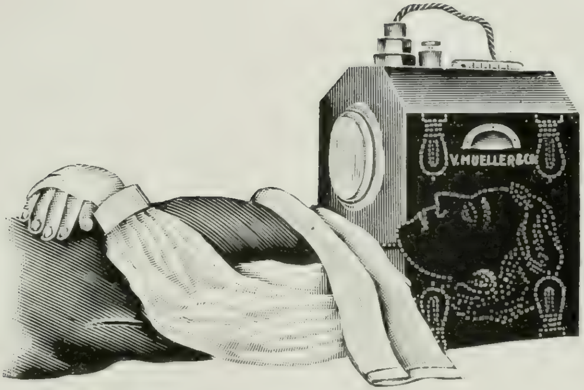
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FIGURE 6.

Headlight bath. A thermometer that does not show in this cut records the temperature. The patient breathes the outside air. Connections not shown.

XLII.

PRIMARY DISEASES OF THE LABYRINTH AS THE RESULT OF FOCAL INFECTION.*

By George E. Shambaugh, M. D.,

Chicago.

Accurate knowledge of the diseases of the internal ear has been very difficult to acquire, owing especially to the difficulty surrounding the study of the pathology of the labyrinth. In recent years a great deal has been done to clear up obscure facts relating to the pathology of those changes in the labyrinth which occur secondary to infection in the middle ear. The pathology, also, in cases of long standing nerve deafness, is now well understood. There still remains a great deal of obscurity regarding the pathologic changes in the labyrinth, especially in cases of primary disease of the internal ear. Here we still find it necessary to piece out our knowledge by analogy from similar conditions occurring elsewhere. Fortunately, our increased knowledge of the physiology of the labyrinth enables us now to interpret much more accurately than heretofore the symptoms arising from disease of the internal ear. Our improved methods for making a functional examination, moreover, enable us to detect more readily disturbances in the function of the labyrinth.

The clinical phenomena observed in these cases of primary involvement of the internal ear are quite definite, although the symptoms vary more or less widely in different cases. The defect in the hearing is always quite characteristic. It begins as a defect in the upper part of the tone scale, while the hearing at the lower part of the tone scale remains normal, even after the defect for the higher notes has become quite extensive. In a few cases the loss of hearing does not begin at the highest part of the tone scale, but consists of a defect more or less circumscribed in the middle of the tone scale. For example, one may find a defect especially marked for the c^4 fork, while the Galton whistle may be heard normally throughout its entire extent. Paracusis Willisii is never a symptom in these cases, and tinnitus aurium, although often present, is rarely, if ever, the annoying symptom which it so frequently is in cases of otosclerosis. In most of the cases both ears become involved sooner or later, and when both are involved the shortening of bone conduction is always present. In one sided involvement the positive Rinné will be changed to a negative in those cases where the defect in hearing is quite marked.

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*Read at the meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City.

Symptoms arising from an involvement of the vestibular part of the internal ear constitute a very important part of the clinical phenomena observed in many of these cases. Primary disease of the internal ear constitutes, in fact, the most frequent cause for the symptom of vertigo. An involvement of the vestibular apparatus in these cases is, moreover, much more common than the occurrence of vertigo would indicate, as we have been able to ascertain since the introduction of tests for examining the function of the semicircular canals.

Another important clinical fact that has been brought to light by the introduction of the vestibular tests is that primary degeneration of the vestibular nerve occurs quite independent of a similar process involving the cochlear nerve. In these cases an occasional attack of vertigo is the only symptom indicating disease of the labyrinth.

We see, therefore, three distinct types of primary degeneration in the labyrinth: one where the cochlea alone is involved, producing nerve deafness and more or less tinnitus; the second, where the vestibule is involved simultaneously with the cochlea, producing, as a rule, occasional attacks of vertigo in addition to the symptoms arising from disease of the cochlea; the third type, where the vestibular nerve alone is affected, and where all symptoms indicating disease of the labyrinth may be absent, except for possible occasional attacks of vertigo.

The progress of this disease differs widely in different cases. Not infrequently there appears to be a gradually increasing degeneration of the parts involved. In many cases progress of the degeneration is accelerated by acute exacerbations. In others an acute attack is apparently followed by a long period of quiescence, when there is no progress in the degeneration—at least none that can be detected by our examinations.

The variations in the symptoms depending on the way in which the disease of the labyrinth progresses is especially striking for those cases where the vestibular part is involved. An insidious degeneration produces no symptoms excepting the gradual development of nerve deafness associated, perhaps, with some tinnitus. Where the degeneration is accelerated by acute exacerbations, this is recognized by an increase in the deafness and the development of a more distinct tinnitus; and where the vestibular part is involved, by the occurrence of distinct attacks of vertigo. The symptoms arising from the vertigo sometimes quite overshadow the other phenomena. When the vertigo is severe enough to cause nausea and vomiting, the cause of the disturbance is often mistaken for some gastric disorder. The actual disturbance produced by the vertigo varies from a slight disturbance of equilibrium to a profound shock, which throws the patient violently to the ground and produces dizziness which does not disappear for several days or even weeks. Where one has an opportunity of examining a case during the attacks of vertigo, he is able to recognize the characteristic vestibular nystagmus.

The question of the diagnosis of labyrinth disease is especially interesting in those cases where the principal symptoms are those resulting from the vertigo, such as nausea and vomiting. Before the introduction of the vestibular tests the diagnosis of a possible labyrinth origin for the vertigo was based on the presence of symptoms of disease of the cochlea—that is, tinnitus and nerve deafness. In those cases where a diagnosis could be made of a degenerative process going on in the cochlea, it seemed fair to assume a probable labyrinth origin for the vertigo, since it is known that primary degeneration in the labyrinth is the most frequent cause for this symptom. It is evident that in those cases where the degenerative process was limited to the vestibular nerve—that is, where the hearing was not affected—the labyrinth origin for the attacks of vertigo would often pass unrecognized. Fifteen years ago I began compiling a series of such cases where the symptoms complained of were those of vertigo and the disturbance caused by the vertigo, such as nausea and vomiting, but where there was no involvement of the cochlea, and where no other cause for the vertigo could be detected. At that time it was necessary to assume a condition which we could not demonstrate—namely, a primary involvement of the vestibular apparatus, independent of any disease of the cochlea. In recent years, since the introduction of the vestibular tests, I have been able to go over a number of these cases and to demonstrate the loss of function in the semicircular canals.

The etiology of primary degeneration of the labyrinth is quite clear in many cases. It is not infrequently a complication of syphilis, both hereditary and acquired. It is observed also as a sequel of the infectious fevers, especially in mumps, typhoid, measles, and scarlet fever. There remain, however, a large percentage of these cases of primary disease of the internal ear where the etiology has not been accounted for. It is in these cases where I have suggested focal infection as a possible cause. This conclusion was based upon the similarity between the manner in which the labyrinth involvement takes place and the involvement of other nerves where focal infection is known to be the cause. When the involvement of the labyrinth is associated with other evidences of systemic infection of focal origin, such as rheumatism or neuritis, one sees little difficulty in assuming a possible focal origin for the neuritis of the eighth nerve. In cases, moreover, where the degeneration of the eighth nerve develops quite independent of any other manifestation of systemic infection, a possible focal origin should not be overlooked, for it is not an infrequent clinical phenomenon to observe a single nerve the seat of persistent neuritis from focal infection.

I will report here briefly a few cases of primary labyrinth disease where focal infection appears to be the probable cause.

The first case is that of a man, aged twenty-eight years, who consulted me in October, 1914. He had been subject to attacks of acute articular rheumatism since the age of ten, and had some joints which at the time of the examination were acutely involved. Evidence of an old endocarditis was also discovered. There was a history of recurring attacks of tonsillitis, and, although the tonsils had been operated, they had been only partially removed. In February, 1917, the patient consulted me because of an attack of vertigo associated with tinnitus. The trouble had begun six days before as a tinnitus in the left ear, which he had noticed first on retiring. He was wakened in the middle of the night because of vertigo and nausea. The severity of the vertigo forced him to remain in bed for twenty-four hours, and at the time when I saw him, six days after the onset, he was still annoyed by the disturbance of equilibrium. He recalled having had previous attacks of tinnitus but not of vertigo. He was still suffering from acute rheumatism in several joints. A careful examination of the hearing failed to detect any defect. There was no spontaneous nystagmus, but the rotation test disclosed a marked depression of function for both the right and left labyrinth. The nystagmus following the rotation to the right was extremely feeble and lasted but twelve seconds, and after rotation to the left was of the same character and lasted eleven seconds.

The second case is that of a man, aged fifty-one years, who consulted me in March, 1914, complaining of defective hearing, which he thought might have been coming on for several years. There was no tinnitus, no paracusis, and no history of deafness in his family. Examination disclosed normal drum membranes. A defect in the hearing for the whispered voice was found in both ears, especially marked for numbers like 66, which was heard only at one foot for either ear. The C fork, 64 d. v., was heard normally for both ears, as was also the c^4. The Galton whistle (Edelmann) was heard at 4 in the right ear and at 19 in the left.

The patient returned to the city again December 10, 1914, when he complained of a high-pitched continuous tinnitus which had developed during an actue head cold one month before. There was a distinct increase in the defect for the Galton whistle, especially in the left ear. The faucial tonsils were somewhat enlarged and distinctly congested. Cheesy plugs were expressed from both tonsils. There was no accessory sinus trouble, and no infection about the teeth. In April, 1915, the patient discovered that he was running a temperature ranging as high as 100° and 101° each day. At one time it registered 103°. Examination of the blood showed a leucocytosis of 14,000. During this time he developed a severe persistent arthritis in the instep of the left foot, which came on spontaneously and was not the result of traumatism. There were no other rheumatic symptoms. This condition continued without abatement until June, when the tonsils were removed, after which there was a gradual subsidence of the temperature and disappearance of the leucocytosis.

The patient was seen again December 6, 1916, complaining of increasing annoyance from the tinnitus in the left ear and from beginning tinnitus in the right. His general condition at this time was good, and the arthritis had practically disappeared. The functional examination of the ears was carefully gone over. There was a distinct shortening of bone conduction, and the Rinné was positive in both ears. The Galton whistle was heard at 6 in the right and 25 in the left ear. There had been at no time any symptom of vertigo, yet the nystagmus after rotation to the right lasted but nine seconds, and after rotation to the left but six seconds, and was very weak. The past-pointing after rotation to both the right and left was still present, but very slight.

In this case the chief symptom was the insidious development of nerve deafness, which became quite extensive in the left ear. Tinnitus developed after an acute infection only after the condition had been present several years. At no time was there any vertigo, yet the examination disclosed a very marked degeneration of the vestibular nerve in each ear. The patient's general condition remained good except for a period of several months in 1915, when he was running a temperature. There was no history of syphilis, and the Wassermann test was entirely negative. The patient has suffered from no infectious fever, but he did suffer from distinct evidence of a systemic infection, as shown by the protracted rise in temperature, the leucocytosis and the arthritis. It seems logical to assume that the same cause was responsible for the degenerative neuritis which involved both branches of the eighth nerve.

A third case is one where the neuritis of the eighth nerve followed distinctly from an attack of acute tonsillitis. The case is briefly as follows: Mrs. H., aged thirty-four years, consulted me May 15, 1917, especially because of vertigo and deafness. She gave the following history of the onset of her trouble: Two weeks ago she contracted a severe tonsillitis on the left side, which developed with white patches. No cultures appear to have been made. She suffered from severe headache for three or four days During this time she developed facial paralysis on the left side and huskiness of the voice, which examination showed was due to paralysis of the left vocal cord. At the same time she began to suffer from slight tinnitus in the left ear, which resembled the noise of a sea shell and was associated with a defect in the hearing and intense vertigo, with nausea and vomiting.

When she consulted me I found the facial paralysis was still practically complete on the left side, and the left vocal cord was immovable. Drum membranes were quite normal. Transillumination of the mastoid showed normal condition both sides. Functional examination showed the hearing in the right ear to be entirely normal. In the left ear the whispered voice was heard only close to the ear, and it might easily have been perceived by the opposite ear. The hearing for the C fork. 64 d. v., was only slightly shortened in the left ear. The Weber was lateralized to the right. The Rinné test was positive in both ears. The hearing for the c^4 fork was only slightly shortened in the left ear, while the hearing for the Galton whistle was but slightly shortened at the upper limit. A striking peculiarity in the hearing of the left ear for all the tuning forks was that they were heard distinctly at least an octave higher in the left ear than in the right, and this applied to the Galton whistle as well. The Romberg was positive, the patient falling to the left. There was a spontaneous rotatory nystagmus, which still persisted especially strong on turning the eyes toward the left, but disappeared entirely on looking to the right. Spontaneous pointing showed slight deviation to the left. Nystagmus after rotation to the right was very weak and lasted but seven seconds; to the left was quite strong, but disappeared in about twenty seconds. Caloric stimulation produced no response in the left ear, but normal response in the right ear.

Diagnosis.—Peripheral toxic neuritis, as the result of tonsil infection involving the eighth nerve, the seventh nerve, and responsible for paralysis of the left vocal cord. The case was examined carefully by Dr. Thor Rothstein, a neurologist, who arrived at the same conclusion.

The last case to which I will refer was a Mrs. K., aged sixty-one years, who consulted me in May, 1917, because of roaring tinnitus aurium, deafness and attacks of vertigo, which developed suddenly January last. She has had a number of attacks in which the vertigo is associated with nausea and vomiting. The patient has been subject to recurring tonsillitis for many years. The tonsils were found to be enlarged, congested, crypts filled with cheesy deposits—in other words, the seat of a chronic infection.

In the functional examination the whispered voice was heard normally in the right ear; in the left ear there was a distinct shortening for 66, but not for 18. Weber was not lateralized. Schwabach test showed distinct shortening of bone conduction. The Rinné was positive in both ears. The C fork, 64 d. v., was heard normally in the right ear and only slightly shortened in the left. The same was true for the c^4 fork. The Galton whistle showed a distinct defect at the upper end of the tone scale, involving both ears. Examination of the vestibular apparatus showed spontaneous pointing normal. There was no spontaneous nystagmus when the patient was examined. Rotating the patient to the right developed a nystagmus which was very strong with rapid movements and lasted thirty-two seconds. Rotation to the left developed a nystagmus which was very weak and lasted only sixteen seconds. In this case there is apparently a neuritis involving both sides, but with the cochlear nerve more involved on the left side and the vestibular nerve more involved on the right.

XLIII.

THE STATIC LABYRINTH IN SYPHILIS.*

By Jesse Wright Downey, Jr., M. D.,

Baltimore.

Syphilis, excepting tuberculosis, is perhaps the most prevalent disease with which we as physicians have to deal. Its frequency is startling, its manifestations are legion, and no organ or tissue, from intrauterine life to the grave, is free from the liability of its attack. The discovery and demonstration of the spirocheta pallida, and the Wassermann reaction in the blood and the cerebrospinal fluid, have conclusively proven the syphilitic nature of many diseases which we have heretofore considered and studied as clinical entities; therefore, though we may retain certain names for the sake of classification, we must accept the fact, as one author has stated it, "that these diseases are syphilitis, and always syphilitic."

We know that syphilis of the cerebrospinal system may simulate any disease of the brain and spinal cord substance, their vascular supply and their coverings, and it is only by the examination of the blood or the spinal fluid that we can definitely determine whether or not a central nervous system lesion is syphilitic in origin. A vast amount of laboratory work along this line has shown that brain syphilis is by no means always a late manifestation of the infection, and thereby it has become possible to diagnose lues of the central nervous system long before the appearance of the classic objective signs that we are used to consider as pathognomonic, such as the Argyl-Robertson pupils, the diminished as abolished tendon reflexes, areas of increased sensibility, etc.†

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*Read by invitation before the fiftieth session of the American Otological Society, Atlantic City, May 30, 1917.

†Wile and Stokes: Involvement of Nervous System During the Primary Stage of Syphilis. Journal A. M. A., March 20, 1915, p. 979. Six cases of early syphilis with involvement of nervous system in four.

Collins: Brain Syphilis. Journal A. M. A., July 10, 1915, p. 139 (Vol. LXV, No. 2).

Such signs in fact belong to a later stage of the infection, and indicate that an irreparable destruction of the parenchymatous cells has already taken place; whereas in the early stage of an invasion, it is vascular structures and the meninges which are involved, and though it does not necessarily follow that a basilar meningitis of syphilitic etiology must lead to an involvement of the brain or cord substance, there is always this possibility, which undoubtedly may be the better averted the sooner appropriate treatment is instituted.

The subject of brain syphilis is of prime importance to the otologist, for the reason that the preponderance of evidence in many investigations goes to show that the eighth nerve is more often affected in all stages of syphilis than any other of the cranial nerves; that the lesion is probably a true neuritis of the eighth, its nuclei, its trunk, or its peripheral distributions; and that its involvement is indicative of the luetic invasion of the central nervous system.

For many years we have recognized that certain forms of deafness were due to hereditary syphilis. The first contribution to the subject was probably published by Feichtman[1] in 1857, though the classic paper of Hutchison and Jackson[2] may be considered the pioneer work, especially as these authors correctly defined the nerve deafness which is characteristic.*

It is not only in hereditary syphilis, however, that the auditory nervous mechanism may become involved. Habermann[3] in 1896 reported sixty-six cases of syphilis of the internal ear, of which thirty-four were attacked during the secondaries. Mayer[4] in 1911 reported sixty-five cases of syphilitic internal ear disease, thirty within the first year of the infection, thirteen within from three to ten weeks following the primary sore. Politzer[5] reported one case occurring seven days after the chancre. All these cases, and many more by various authors, were reported before the advent of salvarsan, to the use of which so many cases of deafness has been accredited. It is far from the purpose of this paper to reopen that controversy, for it is now pertty well understood that insufficient treatment with the arsenical preparation may do harm, although the extent of this has undoubtedly been exaggerated. In 1915 W. W. Willcut[6] presented, before the American Medical Association, a report of two hundred and ninety-three syphilitics examined by him in Urbantschitch's Clinic. None had had salvarsan. In fifty-six cases of a month's duration or less he found fifty-two showing the characteristic deafness of syphilis. All had negative Wassermann reactions, but the diagnosis was certified by the demonstration of the spirocheta pallida in the primary sore. G. P. Wintermute[7] reported three cases of acute syphilitic acoustic neuritis before the same session of the American Medical Association (San Francisco, 1915), and gave complete and valuable data concerning the symptoms, course and treatment of these conditions. The cases studied, both by Willcut and Wintermute, are most conclusive, and the bibliographies furnished by these authors are very complete. Evidence is surely not lacking that the eighth nerve is often affected in primary syphilis, nor is proof wanting that it is also often involved in the late syphilides, and that aural symptoms may be one of the first signs in tabes dorsalis and general paresis. Ellis and Swift[8] found deafness of the nerve type in forty per cent of a series of tabetic cases. Shaller[9] found the internal ear involved in sixteen cases out of thirty-five patients with early tabes. Deafness is now mentioned by all neurologists as one of the symptoms often found in the preataxic stage of tabes dorsalis.

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*C. Ernest West has given a complete historical review of the subject in his chapter "Aural Syphilis" in the monumental "System of Syphilis" edited by D'Arcy Powers and J. K. Murphy.

Naturally, there have been many different views respecting the portion of the ear and of the nervous apparatus of the ear that is affected in syphilis. Sexton[10] attempted to show that the deafness in syphilis was due to lesions in the middle ear, but this contention was completely refuted by St. John Roosa.[11] Moos[12] in 1887 found changes in the vestibule, in the organ of Corti, and in the ampullæ of the semicircular canals, but a normal nerve trunk, in a patient with secondary syphilis who had died soon after ear symptoms had arisen; and thus the trend of opinion from then on was to consider the ear lesions of the secondaries as intralabyrinthine.

Deafness occurring in tertiary syphilis was studied by Rosenstein,[13] who found lesions in the nerve stem, in the nuclei, in the roots, and in one case in the roots alone (Ellis and Swift[8]). Mannasse[14] has given a most complete picture of the morbid anatomy found in a patient with tertiary syphilis who had been deaf for one year before death. He found changes in the nervous structures of the cochlea, of the semicircular canals, and also in the nerve trunk. In tabes with deafness Habermann[15] found gray atrophy of both auditory nerves, and atrophy of the nerve endings in the cochlea and in the semicircular canals. The nuclei were intact, the lateral and median acoustic roots much atrophied. Bruehl[16] found degeneration in the nuclear region of the eighth nerve. Mayer[17] studied five cases of general paresis with deafness, and found atrophic changes in all the nervous elements of the ear from the organ of Corti to the medulla. Rosenstein[13] believes that deafness in inherited lues is due, in many instances, to a basal meningitis, though other observers place the lesion within the labyrinth. In 1912, Knick and Zaloziecki[18] examined the spinal fluid in nine cases of early syphilis with deafness, and found a high cell count in all nine and a positive spinal fluid Wassermann in eight. They claimed, therefore, that the deafness in early syphilis is due to a syphilitic meningitis which involves the eighth nerve.

Ellis and Swift[8] corroborated this work in seven cases of their own, and concluded "that all lesions of the eighth nerve are possible manifestations of a disastrous form of a general infection—syphilis of the central nervous system." Willcut[6] believes that the involvement of the cranial nerves in early syphilis is due to a toxic irritation of the nerve sheath and the peripheral end organs. He mentions the eighth, the first and the seventh nerves, and says: "The acoustic, for some unexplainable reason, appears to be the most sensitive of all the cranial nerves." Willcut and many authors seem to overlook the anatomic peculiarity of the eighth nerve being devoid of neurilemma; it is, therefore, all the more liable, on account of its position, to become inflamed during the course of a basal meningitis or from toxic substances circulating in the cerebrospinal fluid. It is hardly possible to doubt the observations of any of the authors quoted, consequently, though we must accept the conclusions that the lesion is to be found in the nerve tissue, we must also accept the fact that the eighth nerve may be attacked centrally, axially or peripherally;

therefore, the careful clinical study of this particular class of patients by the newer and more accurate methods of otologic examination should put us well on the way to that precision in diagnosis which Robert Bing says is wanting—namely, the differentiation between lesions of nerve or its supranuclear tracts and lesions of the percipient structures in the internal ear. It is possible to separately examine both the cochlea branch and the vestibular branch of the eighth nerve, and in the latter tests the phenomenal work of Bárány has been so amplified by the methods and teachings of Randall, Jones, Fisher, Brumm, and others of the "Philadelphia School," that localization of eighth nerve lesions has certainly passed beyond the stage of hypothesis.

It is generally taught that if the cochlea division of the eighth nerve is found to be normal, the socalled static labyrinth and the vestibular nerve will also be normal. In syphilis, however, it is possible for the vestibular branch to become involved at some point along its course, while the cochlea branch may remain normal. That the reverse may be true is admitted by all. In a large percentage of syphilitic cases both divisions of the eighth nerve will be affected and, therefore, both the auditory and the static labyrinth should be tested. It should be remembered that patients with the acoustic neuritis of syphilis may hear the voice surprisingly well and may not complain of deafness until questioned; the tuning forks, therefore, offer the best means of correct diagnosis in these cases.

The typical hearing defect is a shortening of the duration of perception by bone conduction, out of all proportion to the shortening of the duration of perception for the same fork by air conduction; the retention of good hearing for the low forks, with a loss of perception, or a reduction in duration of perception, for sounds of high pitch. All tuning fork tests should be made under the correct law of decrement of the vibrations (Dundas Grant[19]); therefore, we should attempt to determine the shortening in the duration of perception as compared to the normal, and not simply the duration of perception itself.

The Schwabach test is best made with the opposite ear excluded by a "noise apparatus" regulated to that intensity which is just sufficient to prevent contraaudition. If the Rinné is made by separate tests of bone and air conduction, it is not infrequent to find that bone conduction is actually reduced out of all proportion to the reduction of perception by air conduction, as physically impossible as this may seem, and such a reaction is indicative of an acoustic neuritis rather than of an intralabyrinthine lesion. I have found the C^5 (4096 d. v. s.) fork of more use in routine work than the Galton whistle, for the reason that the intensity is so great with the latter instrument that partial defects in high register are difficult to determine.

No matter with what care we may attempt to determine reductions in audition, we must always depend upon purely subjective methods. The tests of the vestibular apparatus are to a large degree objective, and in normal individuals the reactions are surprisingly constant, when the tests are made with precision and with due regard to details. In the rotary tests the after-nystagmus should last for twenty-six seconds, the patient having been turned ten times in twenty seconds. The vertigo should last under the same conditions for twenty-four seconds. Nystagmus should be induced in forty seconds by douching the ear with water at 68° F. These are the normal responses found by I. H. Jones, who has further simplified the interpretation of the reactions by giving us the very easy rule to remember, that the nystagmus, the falling and the past-pointing, are all in the direction of the endolymph flow, with the vertigo in the opposite direction. Dr. Jones and his associates further teach that each semicircular canal has a separate nystagmic and vertiginous tract. They have been able to locate approximately, and to postulate, the situation of these pathways in the brain stem, the cerebellum and the cerebrum; hence, their methods of localization in the eighth nerve lesions, and in intracranial conditions involving the eighth nerve, are the most accurate that we have.

In the last eighteen months I have had the opportunity of making an ear examination of a creditable number of patients with syphilis and syphilitic central nervous system lesions. A number of cases have been examined in the neurologic clinic of the Mercy Hospital, and I am greatly indebted to the neurolgoist-in-charge, Dr. A. C. Gillis, for his cooperation. Many of these patients did not complain of ear symptoms, though it was not difficult to demonstrate an associated aural involvement. A number presented predominant internal ear disturbances, and in a few instances the examination of the ear led to the diagnosis of serious diseases of the central nervous system. Though the auditory function was disturbed in most of the cases, the tests of the static apparatus, made by rotation and cold douching, were of far greater value in absolutely confirming the presence of a lesion in the nervous structures of the ear than were the tests of audition.

Generally speaking, the abnormal response of the static labyrinth in syphilis may indicate a lesion of the vestibular nerve at any point from its nuclei to its peripheral distributions. If we may have "islands of deafness," we likewise may have islands of static inhibition; if these defects are situated neuraxially, the reactions from the vestibular tests may simulate those seen in brain stem, cerebellar or cerebellopontine tumors. As a rule, however, the syphilitic responses indicate involvement of the eighth nerve trunk or its peripheral distributions, and the neuraxial symptoms are likely to be so atypical that they indicate lesion of such widely separated areas that simultaneous involvement of these tracts by a new growth would be impossible, whereas a degeneration of these different pathways is not at all improbable. The most characteristic reaction of syphilitic internal ear disease is a lowering and confusion of all the responses; this may vary from the totally dead labyrinth, giving no responses, to the cases showing all the normal reactions reduced in degree. For example, the nystagmus will last but ten to fifteen seconds or less, and will show fine oscillations in which the slow and quick components cannot be easily differentiated. Vertigo is absent, or it lasts but a few seconds. Falling is not definite. The patient, if tested quickly after rotation, will past-point with one arm and not with the other, or will only past-point for a few inches, or will past-point incorrectly. The responses may be intensified by increasing the stimulation; thus, nystagmus may be absent after ten turns in twenty seconds, to become evident for some seconds after ten turns in ten seconds. Douching with water at 68° F. will bring on the same reduced or confusing reactions after a much longer period than the normal forty seconds, or it may require much colder water to bring out any response. Furthermore, it is apparently possible in these cases for one semicircular canal of the same ear to be more affected than the others; hence, we may get all normal responses from rotation with the head in the upright position (horizontal canals), and no reactions with the head forward (vertical canals), or vice versa. Such a lesion would have to exist in the crista ampullaris of one canal only, or as a partial degeneration in the nerve trunk itself, before the nystagmic and vertiginous tracts separate.

Increased irritability of the static labyrinth, evidenced by prolonged nystagmus, with marked vertigo and nausea, is a sign which may not be overlooked. Crossed past-pointing, right arm to the left, and left arm to the right, after turning to the right, and vice versa, has been noted, as has complete reversal of the past-pointing, both arms to the left, after turning to the right, and both arms to right after left turning. Variations from time to time in all the responses must be expected, and may be of value in following the progress of retrogression of the syphilitic process. The reactions, in most instances, point to the ear which is the more involved, and as a rule both are implicated.

I have been able to study twenty-eight patients in whom the diagnosis of syphilis has been positively established, and though the number is far too small to be of statistic value, the clinical data obtained has indicated the general signs just enumerated. Certain groupings of these cases, and individual histories, are not without interest. The histories are arranged in two series: first, those patients in whom ear disturbances were the complaints for which the patient sought relief; second, those patients with central nervous system diseases upon whom a routine ear examination was made, irrespective of aural symptoms. For the sake of brevity, a synopsis of the salient features are given, and details are reported in full only when they have seemed essential.

There are twelve patients in the first group, who came complaining of deafness, tinnitus and vertigo, the frequency of the complaints being in the order named. Deafness and tinnitus were usually both present, but vertigo was not voluntarily mentioned, though histories of slight attacks were elicited in five cases. A shortening of ten seconds or more in the duration of perception by bone conduction for the C^2 (512 d. v. s.) tuning fork was the basis for further examination of the internal ear, and in every patient under discussion, both the cochlea and the vestibular functions were found to be impaired. Five of these patients were the victims of hereditary syphilis, four of whom had interstitial keratitis. These five cases show practically all the variations of syphilitic eighth nerve involvement.

Case 1.—Marie P., colored, aged nine years. Admitted December 6, 1916. Hereditary syphilis. Interstitial keratitis in active stage. Positive blood Wassermann. Sudden and practically complete nerve deafness of both ears, of three weeks' duration. Both static labyrinths without response in any particular or from any test. No improvement under vigorous mercurial treatment. When the patient was first seen there was a slight amount of hearing present; this has now been destroyed. The child enjoys being literally whirled in the chair, in any position, and can immediately stand erect without a tremor.

Case 2.—Sydney D., white, aged seventeen years. Hereditary syphilis. Incomplete nerve deafness of three years' duration. Positive blood Wassermann. Static labyrinths markedly affected on both sides, though the right ear is the deafer of the two.

Right turning (head upright): Nystagmus for ten seconds. Vertigo for twenty-five seconds. Past-pointing negative in both arms.

Left turning (head upright): Nystagmus negative. Vertigo for ten seconds. Past-pointing of right arm negative. Left arm to right a few inches.

Both ears negative to cold douching in all positions.

Case 3.—Edith L., white, aged sixteen years. Hereditary syphilis. Incomplete nerve deafness of the luetic type, more marked in right ear. Static labyrinths involved. Blood Wassermann positive.

Right turning (head upright): Nystagmus for twenty-five seconds. Vertigo present, but patient complained more of nausea. Past-pointed with right arm correctly to right. Left arm negative.

Left turning (head upright): Nystagmus for fifteen seconds. Vertigo slight. Both arms failed to past-point.

With the cold caloric, nystagmus could be induced from both the horizontal and the vertical canals of both ears. Nausea more marked than vertigo. Past-pointing all negative, except that left ear stimulation of both the horizontal and the vertical canals caused the left arm to past-point correctly.

Case 4.—Mary B., white, aged seventeen years. Admitted July 11, 1916, complaining of deafness of eighteen months' duration, more marked in right ear. Hearing tests showed incomplete nerve deafness of luetic type.

Right turning (head upright): Nystagmus for eight seconds. Vertigo negative. Past-pointing with right and left arm, three inches to right.

Left turning (head upright): Nystagmus for three seconds. Vertigo negative. Past-pointing with right arm negative; with left arm, three inches to right.

The girl has a brother totally deaf from measles (?). Her teeth are Hutchison in type. Her blood Wassermann was positive.

Case 5.—John M., white, aged ten years. Interstitial keratitis in subacute stage. Deafness complained of. Impossible to make satisfactory hearing tests. Right turning gave nystagmus for fifteen seconds. Left turning did not arouse nystagmus. Blood Wassermann positive. Although the tests are incomplete in this case, there is sufficient evidence to show that the cochlea and the static functions are both disturbed.

In cases 2, 3, 4 and 5, the hearing is not markedly impaired. All these people can hear the conversational voice without much difficulty. The vestibular tests have been of far more value in correctly determining the nervous character of the aural disturbances than have been the tests of the audition.

The other seven cases in this group were patients with acquired lues, from whom it was impossible to obtain a history of the primary lesion, except in two instances. The value of the ear tests in this particular may be demonstrated by the citation of one case.

Case 6.—Katherine H., white, aged seventeen years. Admitted August 1, 1916, complaining of deafness of the right ear, which had started July 6th. The hearing tests were very uncertain, but the duration of perception by bone conduction for the C^2 fork seemed shortened about thirteen seconds in the right ear, and ten seconds in the left.

Right turning (head upright): Nystagmus for twenty-two seconds. Vertigo very slight. Right arm past-pointed to left. Left arm did not past-point.

Left turning (head upright): Nystagmus for seven seconds. Vertigo negative. Right arm did not past-point. Left arm past-pointed to right.

The caloric tests and turning with the head in various positions gave confusing, conflicting and abnormal responses. No history could be obtained from the patient. Her blood Wassermann, however, was positive; and her hearing rapidly improved under antisyphilitic treatment.

The next two cases are of interest from the valuable indication given by the ear examination of serious central nervous system involvement.

Case 7.—Deafness. Optic atrophy. Argyl-Robertson pupils. Negative blood Wassermann. Positive spinal fluid Wassermann. Tabes dorsalis.

Louis S., white, aged forty-four years. Admitted June 12, 1916, complaining of deafness, more marked in the left ear. The duration of perception by bone conduction for the C^2 fork was minus twenty-five seconds; duration of perception by air conduction, minus fifteen seconds. The duration of perception by air conduction for the C fork was minus ten seconds; for the C^5 fork, minus five seconds. The right ear showed a slight reduction in the bone conduction. The patient said that his eyes were also bad, and ophthalmoscopic examination disclosed a beginning primary optic atrophy, more marked in the left eye. Vision right eye, 18/20; left eye, 18/49. Pupils unequal, irregular and sluggish to light. Patellar reflexes absent. The man admitted a primary lesion occurring twenty-five years ago.

Right turning (head upright): Nystagmus for twenty-five seconds. Vertigo and nausea. Right arm did not past-point. Left arm past-pointed to right.

Left turning (head upright): Nystagmus for twenty-five seconds. Vertigo and nausea. Right arm did not past-point. Left arm past-pointed to right.

The diagnosis of tabes was simple enough in this case, and, of course, was not dependent upon the ear examination. After intravenous and intraspinous treatment with neosalvarsan, the eye condition improved slightly, and the responses of the static labyrinth became perfectly normal. Unfortunately, we were only able to follow this case for six months when he disappeared from the clinic, and so far we have not been able to locate him.

Case 8.—Nerve deafness. Marked increase in the duration of the after-nystagmus with nausea and vomiting. Negative blood Wassermann. Strongly positive spinal fluid Wassermann. Tabes dorsalis.

Ella R., colored, aged thirty-eight years. Admitted July, 1916, complaining of deafness of the right ear of four years' duration, of the left ear of a few months' duration. The bone conduction of the right ear for the C^2 fork was negative; the air conduction, minus twenty-five seconds. The C fork was shortened twenty seconds in duration of perception, the C^5 fork could not be heard. The left ear showed bone conduction, C^2 fork, minus twenty seconds; air conduction for the same fork, minus thirteen seconds. The C fork was but slightly shortened, the C^5 fork was normally heard.

Right turning (head upright): Nystagmus for fifty seconds. Vertigo, nausea and vomiting. Past-pointing normally to right with both arms.

Left turning (head upright): Nystagmus for forty-five seconds. Vertigo, nausea and vomiting. Past-pointing normally

The cold caloric brought on the same severe reactions from both ears in all positions of the head. The blood Wassermann was negative.

The woman was put on mercury for a week, and at the end of that time the Wassermann was again negative. She drifted away from the clinic to return in January, 1917, complaining that her hearing was worse, and that she had headache and "stomach pain." Her pupils were now small and sluggish to light, while before they had been normal, and she was strongly urged to have a spinal puncture, but refused. On May 10, 1917, she consented to the examination, and the spinal fluid examination was as follows: Cell count, thirty-one per cubic centimeter. Wassermann strongly positive. Ross Jones and Pandy, positive. Diagnosis, tabes dorsalis.

The four remaining cases in this group are not without interest, but they shall have to be dismissed with mention only. All had an acoustic neuritis of syphilitic origin, with involvement of the static functions.

From the work done at the Mercy Hospital I have selected sixteen cases for study, which may be grouped under the heads of the neurologic diagnosis of cerebral syphilis, tabes dorsalis, and general paresis.

Seven of these patients may be classified as cases of early brain lesions, six of whom came to the hospital with the complaint of severe attacks of vertigo, and the examination of the ear showed the static labyrinth to be definitely involved in five out of six. The patient who did not complain of vertigo had had an involvement of the ninth, tenth and twelfth cranial nerves. His static labyrinths were apparently normal; the tests, however, were not as complete as I should like them to have been, and he did show a definite "island of tone deafness" for the C^3, C^4 and the C^5 forks, although he heard the upper range of the Galton whistle.

One patient was seen the day following a severe attack of vertigo. Certain details of her history are of interest.

Case 9.—Annie M., colored, aged twenty-nine years. Admitted March 1, 1917, complaining of severe headache and vertigo. The night before, she had fallen and had to be carried to bed. She gave a definite history of lues, the primary lesion having occurred in 1910. Examination showed a positive Romberg. Tenderness on palpation over skull. Neurologic diagnosis, syphilitic meningitis. Her ear drums were normal; her hearing tests showed a reduction in the perception by bone conduction of twenty seconds in both ears. All other tuning fork tests apparently normal.

Right turning (head upright): Nystagmus for twenty-three seconds. Vertigo for fifteen seconds. Past-pointing negative with both arms.

Left turning (head upright): Nystagmus for twenty-one seconds. Vertigo uncertain. Past-pointing negative with both arms.

Two days later, right turning gave nystagmus for twenty-one seconds; left turning, nystagmus for twelve seconds. One week later (March 28, 1917), with the patient much improved generally, right turning, nystagmus twenty-two seconds; left turning, nystagmus, fifteen seconds. The patient felt so much better that she did not return to the clinic and could not be located. It would seem, from this case, that the vestibular tests offer a most accurate means of following a process of this kind.

I can only touch upon the findings in the other patients of this group, as anything less than complete histories would be of little value. The presence of a positive Romberg in these patients is suggestive of the aural nature of this sign.

Case 10.—Wm. G. M., white, aged twenty-five years. Admitted July 27, 1916, complaining of severe vertigo of three weeks' duration. Romberg positive. Neurologic symptoms pointing to a cerebellar tumor. Spontaneous vertical nystagmus on looking up. Hearing normal. Vestibular tests indicative of a neuraxial lesion, but exceedingly variable and changing from day to day. The patient denied lues, and his blood was negative to the Wassermann test on two occasions. The spinal fluid showed cell count, forty-two; Wassermann inconclusive; globulin test positive. On August 30, 1916, the patient became suddenly and completely deaf in the right ear. He died September, 1916, and the postmortem examination showed an old syphilitic meningitis with a degenerated gumma in the middle lobe of the cerebellum, extending to both lateral lobes, particularly of the right side, and involving the region of Deiters' nucleus.

Case 11.—Chas. L., white, aged forty-seven years. Admitted August 10, 1916, complaining of sudden and complete deafness of both ears and of severe vertigo of ten days' duration. Romberg positive. Neurologic symptoms indicative of a lesion in the region of the corpora quadrigemina. Spontaneous nystagmus to right. Vestibular tests indicative of a neuraxial lesion, though the responses were uncertain and not of great localizing value. Blood and spinal fluid showed strongly marked positive Wassermann. The man left the hospital, but died in a few weeks. No postmortem obtained.

Case 12.—M. M., white, aged thirty-five years. Admitted July 6, 1916, complaining of vertigo and deafness. Typical acoustic neuritic defect in hearing. Nystagmus endured for forty-five and thirty-five seconds with the head upright, and vertigo and past-pointing were normal. With the head forward, right turning elicited neither nystagmus, vertigo nor past-pointing. On left turning the reactions came, though normally. Positive blood Wassermann.

Case 13.—Louis M., white, aged forty-two years. Admitted November 10, 1916, complaining of vertigo, deafness and tinnitus. Romberg positive. Vestibular tests showed reduced nystagmus from both ears, and no vertigo or past-pointing. Hearing defect of luetic type. Primary lesion in 1913. Positive Wassermann.

Seven cases of tabes dorsalis in various stages have been studied. In four of these both the cochlea and the vestibular branches of the eighth nerve were involved. The cochlea branch alone in one, the vestibular branch alone in two. These patients were not markedly deaf; indeed, a history of hardness of hearing could only be obtained in three of the cases. Four of these patients had primary optic atrophy. Generally speaking, the aural defects were not of wide extent, but they were none the less striking.

Case 14.—Ethel W., colored, aged thirty-five years, with tabes and optic atrophy, showed normal nystagmus in both directions, but on right turning the right arm past-pointed correctly to the right, while the left arm past-pointed to the left. On left turning she past-pointed with the right arm to the right, and with the left arm to the left. This past-pointing error was present in all positions of the head.

Case 15.—Harry T., white, aged thirty-six years, with tabes and optic atrophy, showed nystagmus lasting for twenty seconds in both directions, but on right turning he past-pointed with the right arm to the right, and with the left arm to the left. On left turning the past-pointing of both arms was correctly to the left. The error of the left arm on right turning was present in all positions of the head.

Case 16.—Columbus A., colored, aged fifty years, has been under observation by Dr. Gillis for several years. He presents a cerebellar type of tabes dorsalis with a positive Romberg. His hearing is normal. He has a spontaneous nystagmus, vertical in type, on looking up, and he shows but one abnormal vestibular response—viz., a failure in past-pointing corresponding to a brain stem lesion.

One case of general paresis showed complete reversal of the past-pointing.

Case 17.—Minnie S., white, aged thirty-eight years. Neurologic diagnosis: General paresis. Positive Wassermann in spinal fluid. Hearing normal. Romberg suggestive.

Right turning (head upright): Nystagmus very fine in type, apparently lasting for twenty seconds. Both right and left arms past-point to the left.

Left turning (head upright): Nystagmus the same as in right turning. Both arms past-point to the right.

The error in past-pointing is present in all positions of the head.

Finally, I must beg indulgence for a work of such magnitude from which, as yet, I feel incapable of drawing any but the most general conclusions. Syphilis, as a cause of internal ear diseases, is a factor which may not be considered lightly, or as a rarity; and as the static mechanism, as well as the auditory apparatus is often involved, we as otologists have, in the vestibular tests, a means by which an early perversion of the eighth nerve function may be detected. There probably are no pathognomonic vestibular responses in syphilis, but the very confusing and conflicting reactions are in themselves indicative of multiple lesions, a characteristic of any syphilitic invasion. If syphilitic neuritis of the eighth nerve always points to an involvement of the central nervous system, and in one sense this must be true, then, in all justice to the patients, they should command the combined knowledge of the neurologist, the syphilographer, and the otologist, for Ellis and Swift have shown that the prognosis need not be so grave if treatment is instituted in time.

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XLIV.

SERIAL FROZEN SECTIONS OF THE THORAX FROM A CASE OF ANEURISM OF THE AORTIC ARCH.*

BY

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Philadelphia.

Twenty, or possibly even ten, years ago there would have been little or no justification for presenting this paper before such a gathering as the present one. However, the gradual extension of laryngologic activities into the regions adjacent to the nose and throat have so widened our field of interest that we feel that no apology is called for in presenting this case to you. So frequently do men in our special line of work introduce instruments into the thorax, and so well is the normal anatomy of this region known, that it occurred to us that a graphic presentation of a case in which the usual relations are badly distorted by disease might be of interest and profit.

We will present first a very brief abstract of the patient's history. (The full history of this case can be found in the Transactions of the Association of American Physicians for 1917.)

He was in the Pennsylvania Hospital twice, once in the service of Dr. J. Norman Henry, and once in the service of Dr. Arthur Newlin.

W. F., negro, aged fifty years, stevedore by occupation. Fifteen years previous to his death he had had a double infection, Neisserian and luetic. Two years before his death he noticed a bulging in the upper sternal region, which gradually increased. He had had treatment at various times, including injections of salvarsan, which gave him considerable relief, but in spite of this he continued to grow worse, suffering from paroxysms of coughing and choking of sufficient severity to keep him awake. His main symptoms were dyspnea and pain in the chest and upper extremities. Later in the course of the disease insomnia was marked. He spent most of his terminal time in bed leaning forward, resting his head and elbows on his knees, and during his last days he was kept more or less constantly under the influence of large doses of morphin.

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*Read before the Thirty-ninth Annual Congress of the American Laryngological Association, held at Atlantic City, May 28, 29 and 30, 1917.

Six days before his death 660 cubic centimeters of clear amber fluid were withdrawn from his left pleural cavity. For the last two months of his life there was a softened area at the apex of the aneurismal protrusion from which a thin sanguineous fluid constantly exuded.

Finally one morning he was seized with a violent hemorrhage from the mouth and nose and expired in two or three minutes. Death was believed to have been due to rupture of the aneurism into a bronchus.

After death his body was secured and brought to the laboratory of anatomy of the University of Pennsylvania. Instead of performing an ordinary autopsy, permission was obtained to do what he wished, and we therefore had the body injected with formaldehyd solution and frozen. Then the thorax was isolated from the head and neck, arms and lower abdomen, and cut into serial sagittal sections about two inches thick.

Figure 1 is a photograph of the patient taken shortly before his death. It shows clearly not only the aneurism itself, but also the eroded area through which bloody serum was constantly seeping.

Figure 2 is a section, viewed from the left, cut approximately through the middle of the clavicle. It shows nothing of the aneurism itself, but a number of the pressure effects of the latter. The lung is seen to be collapsed and the pleural cavity filled with coagulated and frozen serum. The stomach is almost filled with clotted blood, and posterior to it can be seen a portion of the left lobe of the liver. The diaphragm is depressed, more so posteriorly over the liver than anteriorly over the stomach, the dome being practically obliterated.

Figure 3 is a view of the same section as Figure 2, after the pleural sac had been cleared of serum. This shows very well the atelectatic condition of the lung and brings into plain view the pericardium and the upper surface of the diaphragm. On the anterior surface of the upper lobe is a long fibrous scar, which upon section was found to extend into the lung tissue to the depth of almost one centimeter. The lung above this scar and also at the extreme upper portion of the left apex was adherent to the costal pleura. The aneurismal protrusion is shown very clearly in this figure as well as in Figure 2.

Figure 4 is a section cut just lateral to the left margin of the sternum, and the section is viewed from the right; this is the right side of the slab of tissue whose left side is shown in Figures 2 and 3. The aneurism and the pleural sac have been cleaned of serum and clots, and the vessels and air tubes of the lung of their contents.

In this section the aneurism comes into view for the first time. It is apparently of an hour-glass form and has burrowed through the chest wall above and below the second costal cartilage. The condition, however, is only apparent, as probably the sac eroded forward first through the sternum and then advanced laterally, destroying first the soft tissues and later the bone and cartilage. It is interesting to note the thinness of the anterior wall of the aneurism, consisting as it does only of skin and fascia. In the recent condition the whole of the hour-glass sac was solidly filled with laminated clot. Just below the posterior part of the sac is a small diverticulum, and anterior to this is a calcified lymph node.

The upper and lower parts of the interlobar pulmonary fissure are visible, and separated by it can be seen the collapsed lobes of the left lung. The atelectatic condition of the lung causes it to appear to be much more closely studded with vessels and bronchi than is really the case.

The heart comes into this section in the shape of the lower part of the left ventricle. The base of the heart has been pushed far downward by the aneurism, and hence the columnæ carneæ run much more horizontally than is normal. One papillary muscle shows very plainly.

Figure 5 is a view of the left surface of the next section. If the slices of tissue were placed together in proper order this surface would be in contact with that shown in Figure 4. It differs from Figure 4 in that the various cavities were not cleansed of their contents, the clot being allowed to remain in the aneurism and the coagulated frozen serum in the pleural cavity.

The aneurism is well shown, filled in this (its left lateral) portion with a firmly organized laminated clot. After thawing out, the clot was found to have the consistency of a stiffened sponge, and its interstices were filled with blood and serum. It is obvious at a glance how futile would have been an attempt to have treated this case by wiring.

When the pleural outlines are compared with those of the lung, the great degree of pulmonary atelectasis present can be well appreciated. The bronchi were filled with purulent secretion, and this condition may have helped to account for the patient's occasional rise in temperature.

The pericardial sac in this section was densely calcified.

The left ventricle is shown in this section, and one of the papillary muscles is beautifully depicted. The position of the latter helps to emphasize the abnormally horizontal position of the heart.

Figure 6 depicts a section through the middle of the sternum and spinal cord, the slab of tissues being viewed from the right.

The aneurism here appears in its largest dimensions and it is seen as it really is, one large single sac. It has eroded completely through the sternum, all of the manubrium and the upper part of the gladiolus being completely absorbed. The lower part of the latter projects upward into the aneurism as rough naked bone, apparently not covered by periosteum.

Only a small portion of the sac, the posterior portion, remained open as a blood channel during life. The remainder was filled with clot as described above, although blood and serum did ooze through the interstices of the clot and constantly drip through an opening in the skin on the anterior chest wall.

At the posterior part of the aneurism can be seen a small funicular depression with a tiny opening at the apex. This opening was the point of rupture, and the flattened dark area posterior to it is the compressed left bronchus into which rupture took place. We were very fortunate in having this opening occur right in the plane of section. Above and posterior to the aneurism is the bifurcation of the trachea, which is filled with blood.

There was no direct communication between the sac and the esophagus, and the blood which filled the stomach was first coughed up and then swallowed. This process must have been going on for some time before death, since the latter was very sudden and apparently the result of suffocation.

The flattened mass of tissue between the sac and the vertebral column, and extending from the point of rupture up to the tracheal bifurcation, contains the left bronchus and the esophagus. The trachea was pushed very much to the right by the aneurism, and the left bronchus is seen on its way from this right-sided position back to the left lung. It is compressed between the sac anteriorly, and the esophagus and vertebral column posteriorly.

Many interesting things are shown in the heart. Above and anteriorly, and appearing as a tube-like structure, is the conus arteriosus of the right ventricle. At its posterior end can be seen the pulmonary valve separating the conus from the pulmonary aorta. All of these structures are much compressed and forced downward and backward. There must have been a greatly diminished blood stream going to the lungs, and this, in connection with the putting out of commission of the left lung, would amply account for the extreme dyspnea suffered by the patient. At the posterior part of the heart is the left auricle, in its natural position, but much altered from its normal ovoid outline. It is greatly flattened from before backward, especially at its upper part, where can be seen the flattened ostia of entrance of the two left pulmonary veins. The interference here with the return circulation readily could account for the left-sided hydrothorax.

Anterior to the left auricle can be seen the cavity of the left ventricle, with some of the chordæ tendineæ and columnæ carneæ. The thickness of the left ventricular wall is only apparent, as the section happened to traverse the interventricular septum.

Figure 7, like Figure 6, is a midsternal, midvertebral section, but differs from it in depicting the right side of the thorax, viewed from the left. Most of the points mentioned in describing Figure 6 will apply to this plate.

Two leaflets of the aortic valve are shown at the upper end of the left ventricle, anterior to the left auricle.

Posterior to the left auricle, and easily located by tracing it upward from the stomach, is the lower part of the esophagus. It is seen to be filled with clotted blood. In its upper part it was found to be greatly compressed by the aneurism. In view of this it seems rather odd that dysphagia had not been a more prominent symptom. While the patient had been unable to swallow solid food for some time before his death, liquids gave no great amount of trouble, and certainly the blood which reached the pharynx during the last hours of the patient reached the stomach without difficulty. The lower part of the esophagus, in addition to the stomach, was found after death to contain much blood.

In regard to the site of rupture, it is interesting to note the smooth crater-like character of the aortic intima at this point. There is no tearing of a thin vascular wall, as one might expect, but what appears to be a gradual absorption and final giving way of the thick aorticobronchial tissues at a point where pressure had been most marked and longest continued. The point of rupture was exactly where the column of blood driven into the sac by each contraction of the left ventricle would first impinge upon the aortic wall. At this spot was what appeared to the naked eye as a smooth foramen of communication between the aneurism and the bronchus, and the aneurismal intima and the bronchial epithelium appeared to be continuous structures, which is highly improbable.

Figure 8 depicts a section, viewed from the right, cut just lateral to the sternum, in the same relative plane on the right side as Figures 2 and 3 on the left. This plate shows the gradually decreasing size of the aneurism as the distance from the midline increases, and when compared with Figures 2 and 3, shows that the preponderance of the sac is on the left side, as would be expected. The right bronchus is filled with blood, this lending support to the belief that suffocation was the cause of death, but retains an approximately normal shape and position, instead of being displaced and compressed, as noted on the left side.

Below the bronchus is seen the extreme right end of the left auricle, just medial to the place of entrance of the right pulmonary veins.

The main points of interest in this report we believe to be the following:

1. Photographs of the patient obtained at various periods during life showed successive steps in the growth of the protruding mass.
2. Frozen sections were made of a case which had had careful clinical study, and as a result conditions as they had existed in vivo could be accurately reproduced and deliberately studied after death. Search of the literature reveals the fact that this is the first opportunity of this kind which has arisen and been taken advantage of.
3. The exact site of rupture, and the cause and mode of death, could be determined and depicted with a degree of accuracy, certainty and detail which would be impossible of attainment in an ordinary autopsy.
4. The hydrothorax, the pulmonary atelectasis, and the extreme dyspnea are explained in a manner which by any other method of study would have been much less satisfactory, if at all possible.
5. The last statement is equally true of the anatomic relations. The altered position and general changes in the bronchial tree and other thoracic contents are depicted in such a graphic manner as to show to the laryngologist and internist alike what does take place in the presence of such a lesion as this large aneurism. It also suggests what may take place in some of our future clinical cases in the presence of other disturbers of the normal intrathoracic relations.

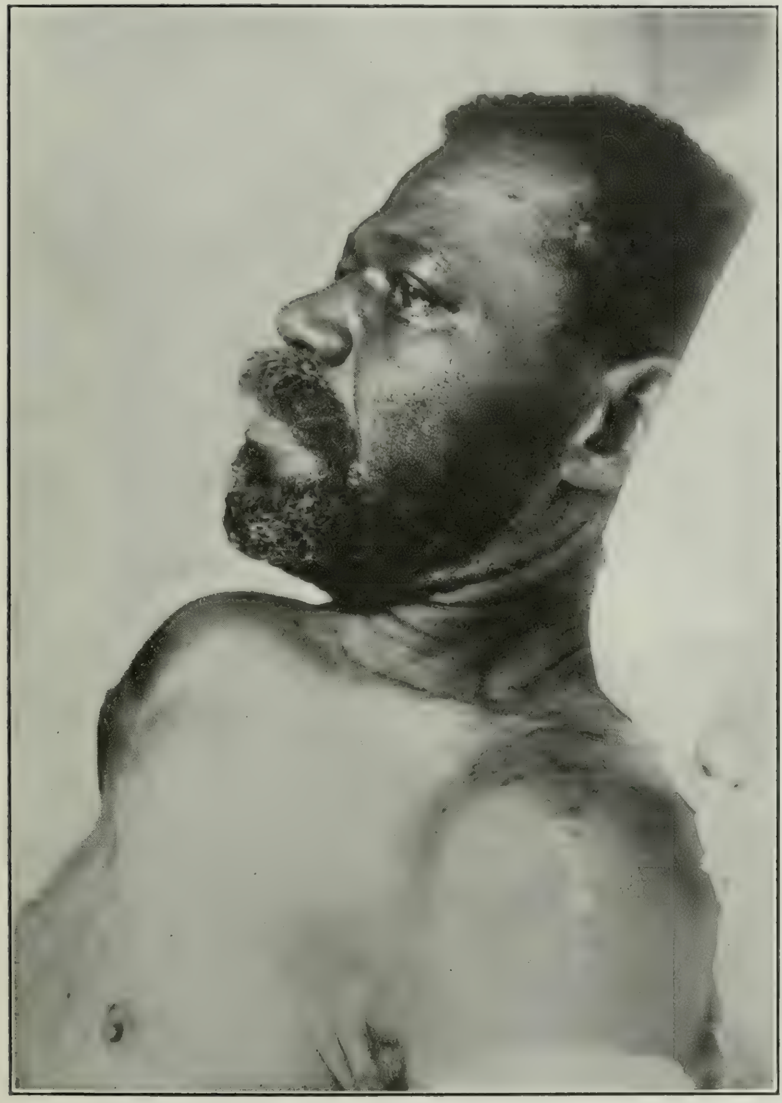

FIGURE 1.

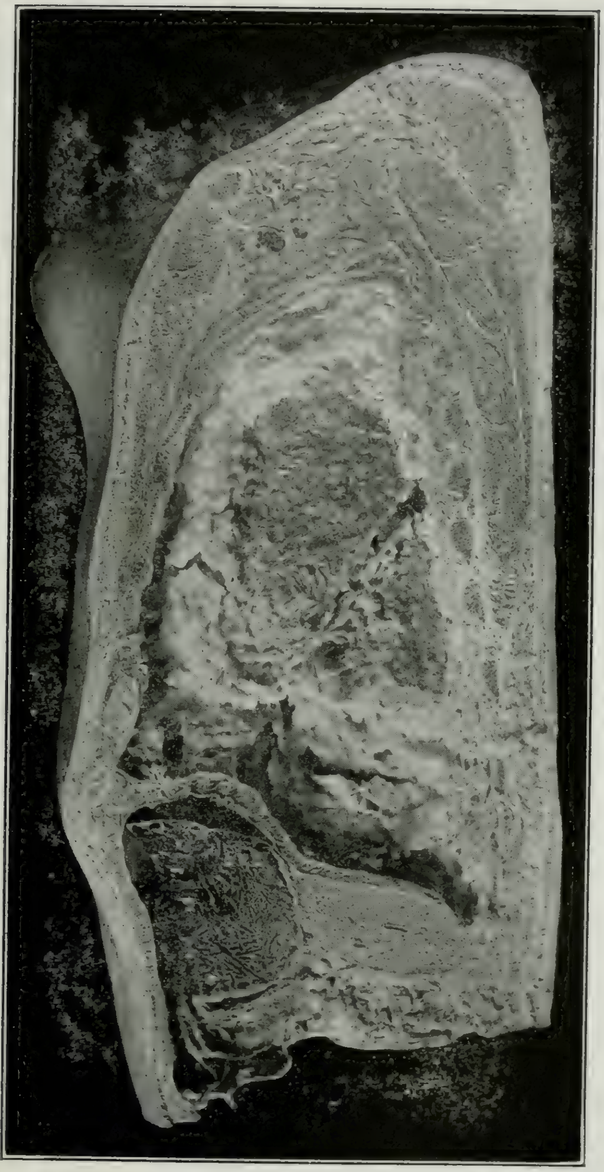

FIGURE 2.

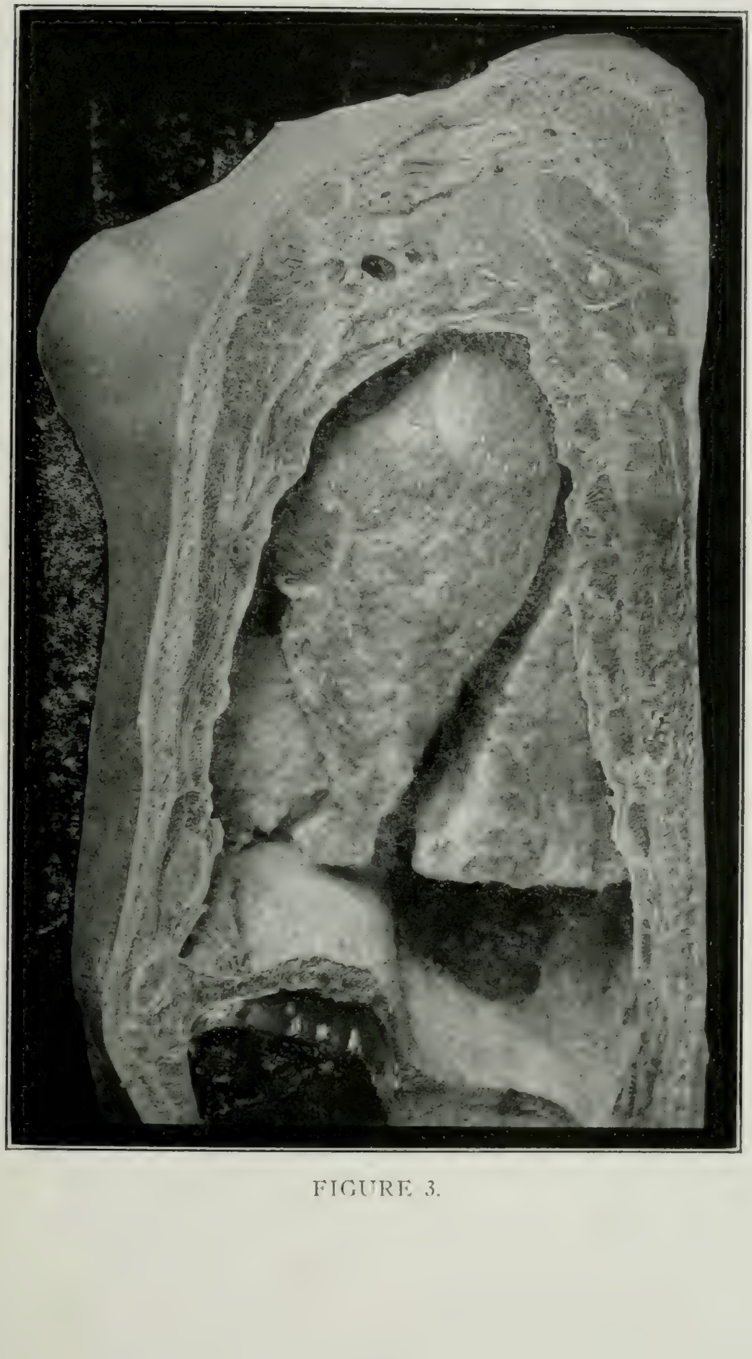

FIGURE 3.

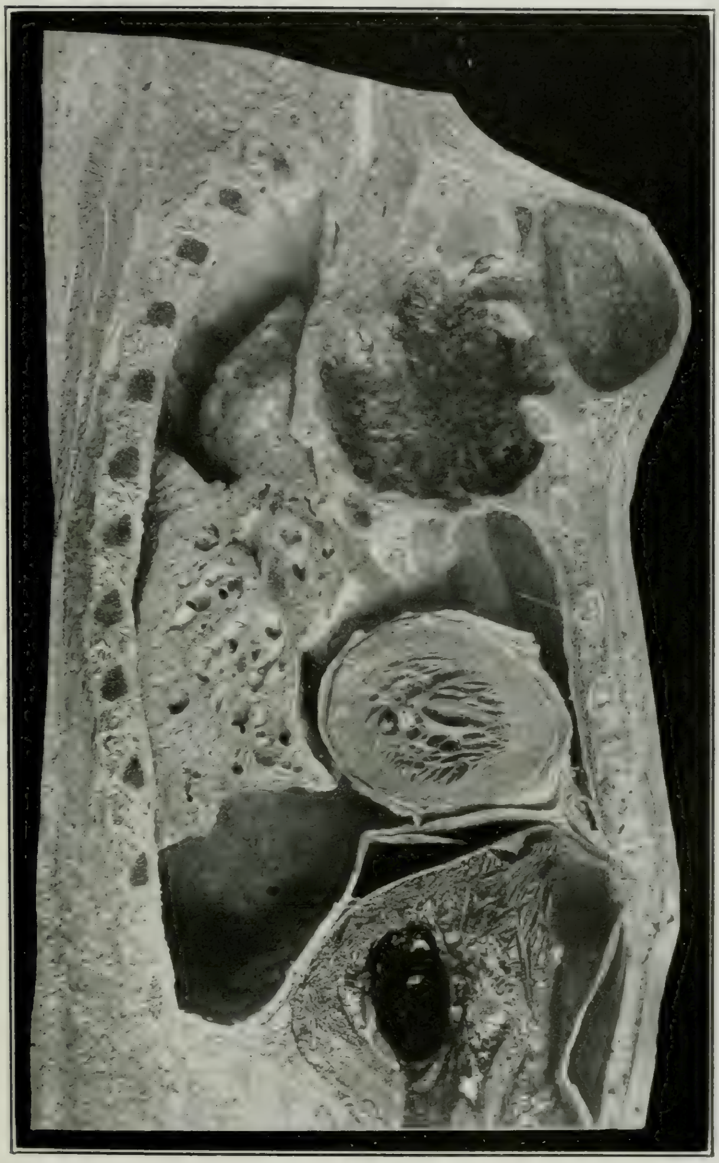

FIGURE 4.

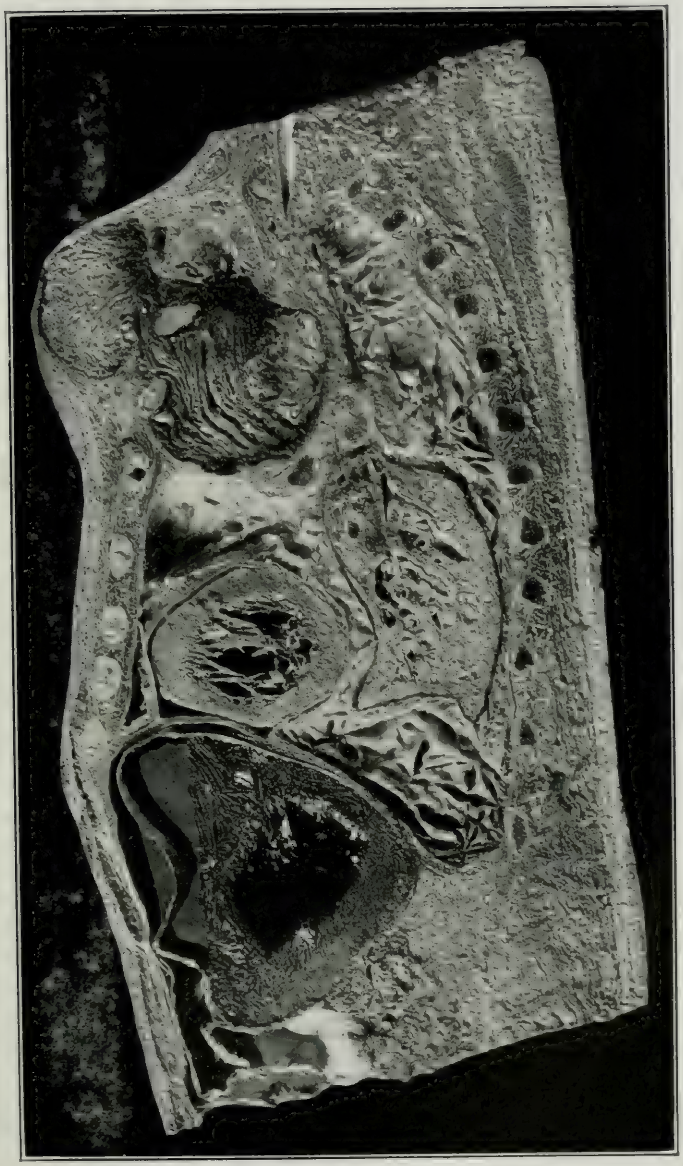

FIGURE 5.

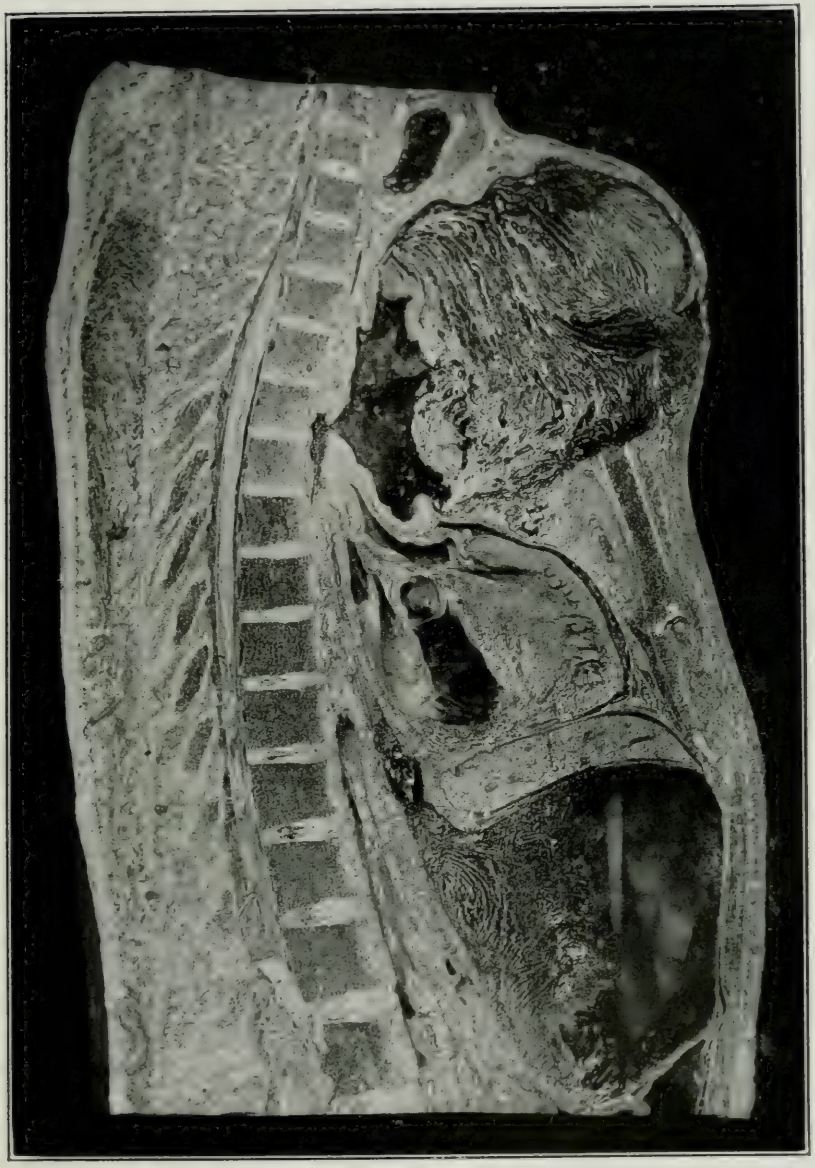

FIGURE 6.

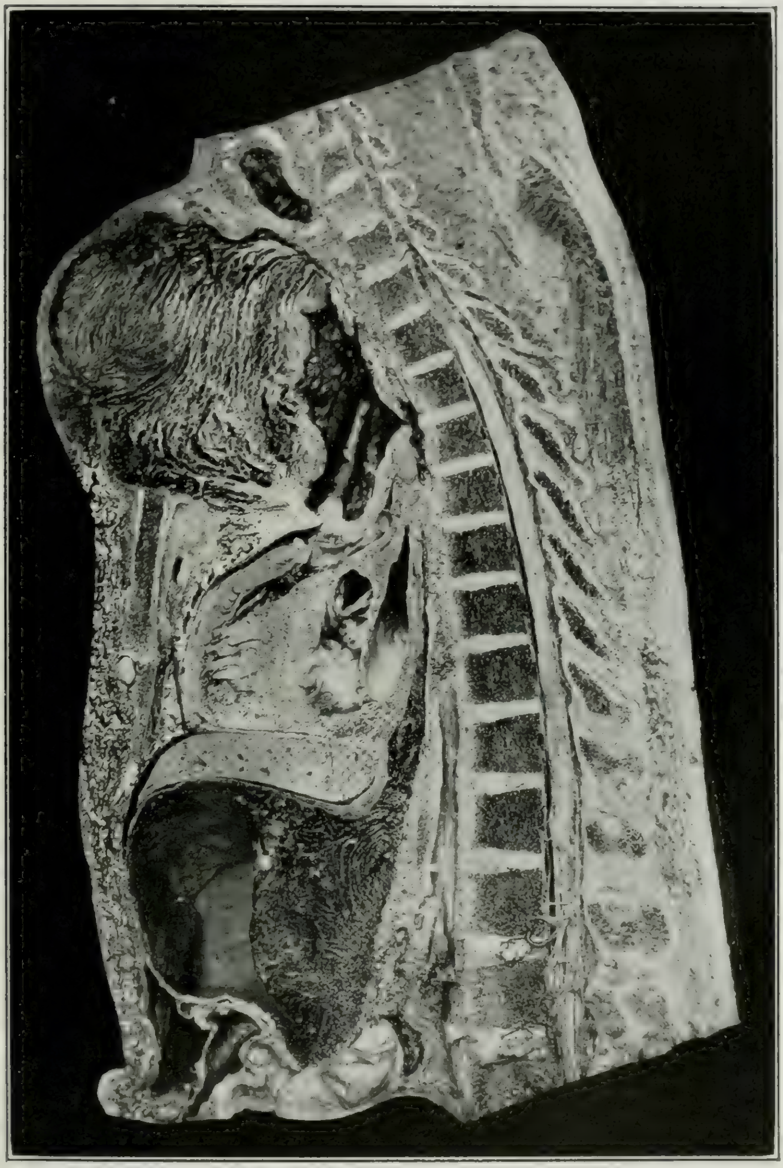

FIGURE 7.

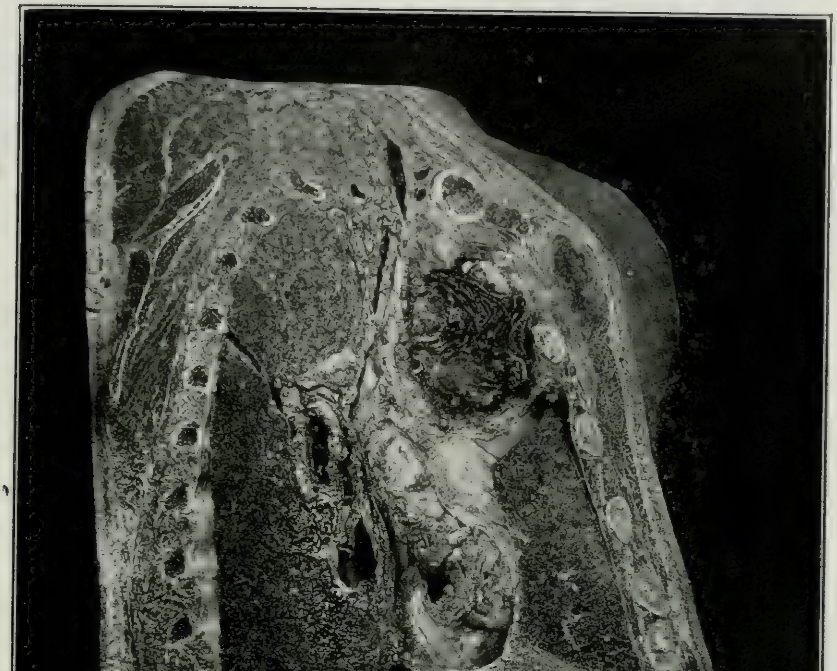

FIGURE 8.

XLV.

THE VALUE OF THE GALVANIC METHOD OF TESTING THE FUNCTIONS OF THE INNER EAR AND EIGHTH NERVE.*

By George W. Mackenzie, M. D.,

Philadelphia.

There are but three known methods of testing the function of the inner ear: the caloric, the rotational, and the galvanic. The first two methods are applicable to the semicircular canals only; the last mentioned is applicable not only to the canals, but also to the cochlea. There is but one method of testing the eighth nerve, and that is the galvanic. Accordingly, the galvanic test has a wider scope of application than either of the other two methods or both combined.

Let us next consider the relative accuracy and value of these three methods as applied to the semicircular canals in the order in which they are mentioned.

First, the caloric. This method has been tried out by numerous investigators and clinicians sufficiently to permit us to draw some definite conclusions. The caloric method is quite reliable when properly conducted and under favorable circumstances—when, at most, it can tell us that the labyrinth is either completely out of function or that it is not. If there is diminution of function, short of complete loss, we have no way of determining the fact by the caloric test, nor are we able to determine the presence or absence of an irritative process within the labyrinth; for instance, congestion, which so often precedes actual inflammation.

The caloric is by no means a quantitative test, in spite of the attempts of Ruttin to make it such. It can give us no information as to the amount of function or hyperfunction that may be present in the labyrinth. Its only value, therefore, as previously stated, is to determine whether the inner ear or, rather, the semicircular canals, are completely out of function or not. In making the caloric test the technic as practiced by the average clinician is faulty, in consequence of which errors of judgment may happen.

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*Read before the American Laryngological, Rhinological and Otological Society, at White Sulphur Springs, 1916.

The average otologist selects cold water and syringes the affected ear with the patient's head upright, when he expects to obtain a rotatory or mixed rotatory and horizontal nystagmus to the opposite side if the inner ear is intact, and no reaction if it is destroyed. This was the method preferred by Bárány, and generally adopted by others. Since most of the cases to which the caloric test is applicable are those of acute labyrinth suppuration, where the patient already manifests a spontaneous nystagmus to the opposite side, it becomes a question of nice judgment to tell whether the cold water has increased the nystagmus or not. To overcome the possibility of mistaken judgment I have suggested the plan of syringing the ear with cold water with the head inclined to the opposite side, with the face forward, so that the ear to be tested is directed toward the zenith. Cold water will then produce a horizontal nystagmus of wide excursions to the affected side if the semicircular canals are reactive, while no effect will be produced if the canals are out of function. With the head in this position there is nothing like the possibility of mistaking the reaction that there is when the head is upright. A substitute for this method is to syringe the ear with head erect and subsequently turn the head to the side position, when a positive reaction may be expected to show itself by the same wide-excursioned horizontal nystagmus to the same side. One advantage the caloric test has over the rotatory test is that it is distinctly a unilateral test. Like the galvanic test, it can be applied to each ear separately.

There are certain circumstances under which the caloric test may not work altogether smoothly; however, with persistent effort they may occasionally be overcome. Among these may be mentioned large polypi in the external canal, large cholesteatoma obstructing the entrance of water into the middle ear cavity, foreign bodies in the canal, atresia (partial or complete, inflammatory or otherwise), excessive congestion, intact membrane, with tympanic cavity filled up with secretion.

etc. Anyone who has had a fair amount of experience can recall instances where one or other of these obstacles has hindered him in making the caloric test.

A partial correction of these conditions, when possible, followed by a second attempt with the caloric test, may result in the finding of a positive reaction when the first effort resulted in a negative reaction. None of these factors act as a hindrance to the galvanic test. Thus far it has been shown that the galvanic test possesses the one advantage claimed for the caloric test, and at the same time carries with it none of the handicaps.

Next the rotational. The one big disadvantage of the rotational test is that it is impossible to eliminate the fellow ear, which is overcome by the caloric and galvanic tests. Its one advantage over the caloric test is that it is a quantitative test; so, too, is the galvanic. The rotational test is a quantitative test of the two ears considered together, while the galvanic is quantitative for an individual ear.

The rotational test is of relatively greater value in testing a single ear when the function of the fellow ear is known to be normal, but when both are affected, either similarly or dissimilarly, allowances must be made accordingly. Even where these unfavorable conditions do not exist, it becomes difficult at times to figure out from the findings the exact conditions in the semicircular canals. It was originally claimed that the after-turning nystagmus in one-sided labyrinth destruction was reduced to eight seconds to the affected side, and to sixteen seconds to the unaffected side. But this is true only in case the semicircular canals on the unaffected side were functionating normally. In some cases we find both ears affected with chronic middle ear suppuration; in other cases, a previous neuritis or other pathologic condition that may have reduced the function in the supposedly unaffected ear, when the figures will be lower.

Let us cite three types of cases which are not uncommon:

Case 1.—Right side. An acute middle ear suppuration, followed by acute labyrinth suppuration. Left side normal. After-turning nystagmus to the right, eight seconds; to the left, sixteen seconds.

Case 2.—Right side. Chronic middle ear suppuration, followed by acute labyrinth suppuration. Left side, chronic middle ear suppuration, in the course of which there has been a secondary plastic inflammation of the internal ear, with partial loss of function in semicircular canals. After-turning nystagmus to the right, five seconds; to the left, ten or eleven seconds.

Case 3.—Right side, chronic middle ear suppuration, followed by acute labyrinth suppuration. Left side, chronic middle ear suppuration, with congestion of labyrinth on the verge of inflammation. After-nystagmus to right, ten seconds; to the left, twenty seconds.

Other cases might be cited. However, it is not necessary for our purpose.

If we are to assume that a case is one of right-sided labyrinth suppuration because the after-nystagmus is eight seconds to the right and sixteen seconds to the left, our assumption is correct only when we can be sure that the left semicircular canals are in normal function. If the function of the left canals is impaired, then the reaction eight and sixteen seconds would be too high, and the probability would be that the function of the right canals is not entirely lost. On the other hand, if there exists hyperfunction on the left side because of a congestion, it would lengthen the after-nystagmus to both sides, in spite of the fact that the right side may be entirely destroyed.

It was because of these very factors that so much confusion existed among authorities in the early days (1906 and 1907), and still exists somewhat to the present day. Another factor that led to confusion on the part of some investigators, and came nearly putting this method of testing the labyrinth into bad repute, was the faulty technic which I had the privilege of pointing out to this society a year ago.

If we were to accept Bárány's exceedingly variable figures for after-turning nystagmus in normal individuals, the rotational test is surely a very unreliable method for examining the function of the semicircular canals.

The rotational method has been merely touched upon, but sufficient has been developed to show that at best it is a quantitative test of semicircular canal function of the two sides taken together, while the caloric method is a qualitative test of semicircular canal function of either side considered separately.

Eight years have passed since the writer first published the results of his investigations with the galvanic method. The figures which were then published, together with the conclusions, have been generally accepted, while Neumann assumed the credit, at least this is the impression to be gathered from certain American textbooks and the hearsay of returning students who have visited Vienna. To the best of my knowledge, Neumann never conducted experiments similar to mine, nor even published anything of a similar nature, nor had any direct or indirect connection with my experiments or publications of the results of those experiments. If wrong in a single statement of these facts, I shall be grateful to the one who points it out to me, and shall make public announcement of it with an apology to Neumann.

Coming back to the subject of the galvanic method of examination of the semicircular canal function, there is very little to add to what I have already published on the subject. During the last eight years where an examination called for a test of semicircular canal function, the galvanic method has never been neglected—and it might be added that there have been numerous calls for the test. Hardly any ear-case that presented itself for treatment escaped the test. It has been tried repeatedly when the patient was too sensitive to submit to the caloric and rotational tests. The average case that balks at the caloric or turning test after the first attempt, consents readily to the galvanic. All other things being equal, this fact alone should give the preference to the galvanic method.

The next question to suggest itself is the value of the galvanic test from the standpoint of accuracy, as compared to the other two methods. It has been pointed out already that the caloric test is quite accurate in selected cases, but may not be in others. Should the examiner fail to recognize certain unfavorable factors (mentioned above), it is possible that he might be led to believe the semicircular canals are out of function when in fact they are not. Acting according to the findings under such circumstances, he might not do what is best for the patient's interest. Such a mistake could not happen with the galvanic test.

If we were to accept Bárány's figures for after-turning nystagmus, we are forced to conclude that the rotational method is to be mentioned only to be condemned. We find Bárány's figures for after-turning nystagmus to vary from one to one hundred and twenty seconds, with marked variations on different days, and marked variations on two sides on the same day. His figures in disease overlap considerably those in health. I am less pessimistic—or, rather, the results of my experience lead me to look more favorably upon the rotational test than it is possible for anyone else to, who accepts Bárány's figures as conclusive.

Up to the present time I have been occupied in pointing out certain limitations and inaccuracies of the caloric and rotational tests; now let us consider the galvanic test from the standpoint of accuracy; its wider application compared with the other two methods having already been referred to when discussing the caloric and turning methods.

In making the galvanic test certain essentials are required: (a) A suitable electrical apparatus with clean contact points. (b) Even more important is an accurate milliamperemeter. (c) A reversing switch under the control of (d) a capable assistant who controls the polarity, the amount of current, and observes the milliamperemeter. (e) Two electrodes, one about two inches square and flat, and the other a small ball electrode (about a quarter inch in diameter, although one-half inch will work satisfactorily). Both electrodes should be wrapped snugly in moist cotton or gauze.

Before starting the test, examination should be made to see if the patient has any spontaneous nystagmus. Its presence or absence should be noted on the patient's record. After soaking the electrodes and the parts to which they are to be applied with warm salt solution, we are ready to begin. The large flat electrode is held by the patient—it does not signify in which hand. The patient's head is tilted slightly backward to permit of better observation of the eyes by the examiner, while he is directed to look straight ahead, which in effect is slightly downward, considering the position of the head inclined backward. The examiner elevates the lid of the eye to be observed with the thumb of the left hand when applying the ball electrode with his right hand to the patient's left ear, and vice versa. To be accurate, I will emphasize the fact that the ball electrode is applied to the tragus, and the tragus is pushed into the external canal. I am anxious to make this clear, since someone recently, in a meeting of the Philadelphia Laryngological Society, claimed he could not get the results he had hoped for with the galvanic test. Upon questioning him he answered that he applied the electrode to the patient's mastoid, after the manner indicated in my book, which evidently he had not read carefully.

In making the test my back is more or less to the instrument. I do not permit myself to know the polarity used. The moment a reaction is noted it is called out to the assistant with the polarity, and the assistant calls back that the polarity is correct and announces the milliamperemeter reading, when a record is made of it. The polarity is switched or not as the assistant sees fit, the important point is that he is not to indicate it in any way to me, the object being to eliminate the possibility of any guessing. Including my published experiments of eight years ago and those conducted since, there have been examined and recorded a few thousand cases. Among these cases are included normals, deafmutes, one-sided labyrinth destructions, acute and latent labyrinth suppurations, old cases of plastic labyrinthitis, unilateral and bilateral, recent as well as old cases of meningitis, some with complete and others with partial loss of function, hemorrhage into the labyrinth, fistulas of the labyrinth, internal auditory canal tumors, herpes zoster, multiple neuritis of the cranial nerves, early and late syphilis of internal ear and nerves, skull fractures, gunshot wound of the ear, cases with middle ear catarrh, others with middle ear suppuration with hyperemia of labyrinth. Many of these cases have been reported singly from time to time during the last eight years.

As a result of these experiments I am led to conclude that the normal reaction from the combined inner ear and nerve is:

Right Ear: Kathode 4 ma., nystagmus to the right. Anode 4 ma., nystagmus to the left.

Left Ear: Kathode 4 ma., nystagmus to the left. Anode 4 ma., nystagmus to the right.

That is to say, the kathodal reaction of one side balances with the kathodal reaction of the opposite side.

That the anodal reaction of one side balances with the anodal reaction of the opposite side.

That the kathodal reaction balances with the anodal reaction of the same side, and that the average reaction for normal individuals requires a current strength of about four milliamperes.

In the case of recent labyrinth suppuration, say in the first week, right side affected and left side normal, we have:

Right Ear: Kathode 7 ma., diminishes the existing nystagmus to the left, while kathode 8½ ma., produces nystagmus to the right Anode 1 ma., increases the nystagmus to the left.

Left Ear: Kathode 1 ma., increases the existing nystagmus to left. Anode 7 or 8 ma., arrests the nystagmus to left, or may throw it slightly to the right.

The explanation of this reaction I have given elsewhere, but will repeat it. Since the normal tonus is equivalent to four milliamperes, it takes four milliamperes suppression (with the anode) to permit the normal tonus of the untouched side to pull the nystagmus over to it's side. For instance, in the case just cited above it takes from seven to eight and one-half milliamperes on the right side to overcome four milliamperes loss of tonus from labyrinth suppuration. Since the tonus of one side must excel the antagonist by four milliamperes, it takes four plus four milliamperes, or eight milliamperes. Since the right labyrinth is destroyed by suppuration, the reaction with one milliampere anode applied to the right side must have exercised its influence on the nerve, showing that the nerve is reactive for some time after the labyrinth is destroyed.

At a later date, which varies from a few weeks to several months, the galvanic reaction changes, and we find:

Right Ear: Kathode—10, 12, 20 milliamperes, or even more, in fact, using the strongest current the patient can stand, produces no reaction. Anode—with the strongest current there is no reaction.

Left Ear: Kathode 1 ma., produces a distinct nystagmus to the left, or it will increase the nystagmus where it was present spontaneously. Anode 8 ma., occasionally causes a spontaneous nystagmus to the left to disappear, otherwise there is no reaction. Twenty milliamperes or more will not produce a nystagmus to the right.

The reaction just cited corresponds to the findings of a total loss of function on the right side that occurs after destruction of the nerve following neuritis or tumor of the internal auditory canal, so that we may conclude, with some feeling of certainty, that all cases of labyrinth destruction, whether from suppuration or otherwise, is followed eventually by secondary degeneration of the nerve.

Another reaction which I have found and recently reported to the Philadelphia Laryngological Society corresponds to a case of eighth nerve neuritis of mild degree in which the reactibility of the affected nerve is excessive. For instance, take a case of right sided neuritis with hypersensitiveness.

Right side: Kathode 1 or 2 ma., nystagmus to the right. Anode 6 ma., nystagmus to the left.

Left side: Kathode 6 ma., nystagmus to the left. Anode 1 or 2 ma., nystagmus to the right.

The explanation is that the right nerve was affected with a pathologic irritation which required less than the normal kathodal stimulus to throw a distinct nystagmus to that side. Whereas, because of the hyperirritability of the affected nerve, it took more than the usual current strength to suppress the total tonus with the anode and throw the nystagmus to the opposite side. Further report of this case will be reported later.

In cases of labyrinth congestion which occasionally accompanies acute middle ear suppuration with membrane intact, we may find a similar reaction, when it becomes a clear indication to operate promptly for the relief of pressure because of the danger of a supervening labyrinth suppuration.

The case of neuritis referred to above developed a more aggravated form later, when the reaction changed entirely, and with it the spontaneous nystagmus jumped to the opposite side. The value of the galvanic test in this case was to point out the fact that the case was getting worse instead of improving. I might have been able to ascertain this fact by other methods,but they would have been more painful and less accurate. Fortunately, in the treatment of eighth nerve neuritis we are more often able to record a change in the opposite direction, indicating improvement and eventually restoration of function. This favorable turn occurs quite frequently in cases of syphilitic affections of the inner ear and nerve during the late secondary or early tertiary stage.

My first inclination in presenting the subject of the galvanic method of examining the function of the inner ear and eighth nerve before this society was to consider the subject exhaustively, listing experiments and classifying cases. Later it occurred to me that such a paper would tend less toward creating an interest in the subject than would one that should be a summary of conclusions reached from a fair experience with this and other tests of the labyrinthine functions, hoping thereby to invite criticism which would tend more certainly to bring out points of interest and more vividly than I could hope to do otherwise.

CONCLUSIONS.

1. That the galvanic test is more accurate than either the caloric or rotational test in determining the function of the semicircular canals.

2. Besides being more accurate, the galvanic test is the least annoying to the patient, the most sensitive, and the easiest to control of the labyrinthine tests.

3. That the galvanic test can be applied as a unilateral test of semicircular canal function in all sorts of pathologic conditions, which is an advantage that cannot be conceded to the caloric.

4. That the galvanic test is applicable to cases of suspected unilateral labyrinth suppuration with obstruction in the external canal where the caloric may fail us.

5. That the galvanic test is a unilateral quantitative test of semicircular canal function, which is a distinct advantage over the rotational test, which at best is a bilateral test.

6. That the galvanic test is the only one we possess for testing the function of the eighth nerve in cases of neuritis or cases of secondary degeneration following destruction of the inner ear.

7. That the galvanic test is the only one we possess whereby we can make a differential diagnosis between labyrinth destruction, pure and simple, and eighth nerve neuritis.

8. That the galvanic test is the only test available for determining the progress of eighth nerve neuritis, whether favorable or unfavorable.

In pointing out the greater applicability and value of the galvanic test as compared to the other tests, I do not wish to be misunderstood as condemning in any way the other methods; on the contrary, I find them of considerable value and use them in every instance where it is possible. My intention is rather to show that the galvanic test is, from every viewpoint, the best, and is the one of choice where a single method is to be used.

Concerning opening and closing nystagmus, nothing has been said in this paper, for the reason that it is of secondary importance and does not cover the object of my paper. It would therefore be redundant.

No reference was made to the fistula test, since it does not belong to the class of tests for comparison. It is applicable to a more limited field, and really deserves a separate consideration.

The spirit that prompts the writing of this paper is to awaken interest in the subect, to invite criticism and to urge the more general use of this method, for no examination of the labyrinth can be considered complete where the galvanic test has been omitted.

XLVI.

HEMORRHAGIC MASTOIDITIS.*

By G. W. Boot, M. D.,

Chicago.

Case 1.—A. B., aged seventy-eight years. Left ear began to ache four days ago. Three days ago free paracentesis was done, followed by a profuse discharge of bloody serum ever since. Cultures and smears gave streptococcus. Temperature normal. Yesterday the mastoid became very tender. No edema. No fever. Leucocyte count, 9500. Seen in consultation by Dr. Shambaugh, who agreed with the diagnosis of mastoiditis, after transilluminating and finding the mastoid dark to transillumination. Operation January 7th, at the Evanston Hospital, under local anesthesia, using one per cent novacain plus adrenalin chlorid. This was done on account of the patient's age and kidney condition. He had had a chronic nephritis for a long time before developing the ear trouble. Good anesthesia was obtained. All the mastoid cells were found filled with dark clots. The mastoid was large and very cellular. All the cells, including the antrum, were opened. Only traces of pus were found in the antrum. The sinus was not exposed. The wound was packed with iodoform gauze and partly closed with clips. Condition at the close of operation excellent. Patient sat up and called for something to eat and drink. There was very little bleeding during the operation.

January 8th, noon. Temperature, 102.5°; pulse, 100. Leucocyte count, 17,600.

January 9th. Patient had a good night. 8 a. m., temperature, 98.4°; pulse, 110. 10 a. m., temperature, 101.8°; pulse, 96. Leucocyte count, 26,500. 10 p. m., temperature, 103°. Red and edematous around wound. Ten cubic centimeters of antistreptococcus serum given.

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*Read before the Chicago Laryngological and Otological Society, March 20, 1917.

January 10th. Temperature, 101.6°. Well marked facial erysipelas present. Ten cubic centimeters of serum given. Erythematous surface painted with tincture iodin.

January 11th, 9 a. m. Temperature, 99°; pulse, 88. Erysipelas has spread over face above mouth, including right auricle. Left ear is clearing up. Is better this morning. In the afternoon his temperature was 98.6°; pulse, 88, slightly irregular. Looks used up. Has not eaten today. Says his stomach is acid.

January 15th. Complains of pain in the middle of the outer surface of the left leg. There is a reddened swollen area here that measures about six by ten centimeters. This was painted with tincture iodin. Temperature has risen to 99.2°. Tongue is red, dry and fissured.

January 17th. Temperature normal. Reddened area on leg has gone, but left ankle is red, hot, swollen and painful. Mastoid wound is not granulating, but has considerable grayish sloughing connective tissue exposed in it. Facial erysipelas is all gone.

January 20th. Cellulitis of left leg is present. Aspiration failed to find pus. Leucocytosis of 30,000, of which ninety per cent are polynuclears. Boric acid compresses ordered applied to the leg.

January 22d. Small abscess over lower right orbital margin opened and pus evacuated. Abscess over left external malleolus opened and drained. Hot boric compresses applied.

January 23d. Temperature, 98.6°. Very weak and listless. Tongue covered with sordes. Mastoid wound irrigated. Abscess at ankle irrigated. Not so tender in left popliteal space as he has been.

January 24th. More tender in calf of left leg. Mentality poor. Was delirious all night.

January 27th. Patient appears a little better. The leg is not as much swollen, and his mentality is a little better. His tongue also looks better. He is taking liquid peptonoids.

February 2d. Patient developed another attack of facial erysipelas yesterday and died this morning.

Case 2.—This case I am allowed to report through the courtesy of Dr. Joseph C. Beck, in whose service it occurred at Cook County Hospital. W. G., admitted to Ward 20, December 4, 1916. Jewish sailor, aged twenty-seven years, single. Patient entered the hospital on November 16, 1916, with an admitting room diagnosis of probable renal tuberculosis. His complaint was of pain below both knees, in the region of the right hip, and in chest and shoulder.

Onset and Course.—Patient's illness began seventeen days ago with sharp pain over both tibiæ just below knees. Next day he had a similar pain in both hips, while the pain in the knees improved. Soon he had pains shooting up to the chest, under the ribs and to both shoulders. These would last two or three seconds at a time. He has not been nauseated.

Past History.—Never sick. Had gonorrhea seven or eight years ago. Had a sore which a doctor called a soft chancre eight or nine months ago.

Family History.—Father and mother alive and well. One sister dead at birth. Nine brothers and sisters alive and well.

Habits.—Formerly a moderate drinker. Does not use tobacco.

Physical Examination.—Patient is well developed, but acts as if he were in considerable pain. Scalp, ears and nose negative. Pupils equal and regular, reacting to both light and accommodation. There is pyorrhea. The gums are sore and the teeth carious. The pharynx is injected and the tonsils enlarged. The postcervical glands are enlarged. Chest is normal. There is tenderness to the right of the umbilicus, over the gall bladder, and in the right lumbar region. The upper extremities are normal. There is tenderness over the left ilium and behind the left hip joint. There is a slight nodular swelling over heads of both tibiæ. Crepitus may be felt here.

Blood Pressure.—Systolic, 118; diastolic, 80. Wassermann plus four.

Diagnosis.—Luetic periostitis, pleuritis.

November 28th. Left membrana tympani is red and bulging. Paracentesis done under local anesthesia. Much pus found. Shortly afterwards patient began to moan and seemed to lose consciousness.. An hour later he was better, but did not remember the attack.

November 29th. Foul discharge from left ear. Tender over mastoid.

December 1st. Patient is moaning constantly. Complains of pain in left mastoid.

December 2d. Left mastoid tenderness is extreme. No edema or bulging. Ear is draining freely.

December 4th. No induration over mastoid. Stuporous. Answers questions with difficulty. Perspires profusely. Pulse slow, full and regular. Temperature, 100°.

The X-ray report on November 28th was as follows: The mastoid cells are very extensive on both sides. The right side is normal in appearance, the left nearly so, being very slightly less clear in outline in the central region. The peripheral cells are clear. Another X-ray report, dated December 5th, states that the "left mastoid region appears to be typically normal. The right mastoid slightly less clear than the left, but I am unable to state that this indicates pathologic change." Patient appears quite toxic. There is profuse discharge from the right ear. The drum is red and there is considerable tenderness on pressure over the mastoid. There is no rigidity of the neck and no Kernig. The knee jerks are lively. The pupils react normally and are equal. There is no swelling of the optic discs. There are no focal symptoms.

A mastoid operation was done on this date. The mastoid was found to be large with numerous cells of moderate size. All the cells were found filled with very dark red, almost black, blood clots. There was a small amount of pus in the antrum and some in the larger cells. Cultures from the mastoid gave a streptococcus. Patient made an uneventful recovery, and was discharged January 3d.

I have found but one case of hemorrhagic mastoiditis reported in the literature—a case reported by Loeb in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY for September, 1915.

Loeb's patient was a nurse who had had no previous ear trouble. She was taken with an acute suppuration of the middle ear which discharged spontaneously inside of twenty-four hours of the onset a large quantity of serosanguinolent fluid containing a diplostreptococcus. The mastoid operation was done inside of four days from the onset of the disease, and the mastoid cells were found filled with dark blood clots. No pus or granulation tissue was found. Cultures from the mastoid were fatal to rabbits. The patient recovered.

The conditions in these three patients were much alike.

The onset was severe. The discharge was a bloody serum and was profuse. The mastoid tenderness was very marked. The temperature was moderate. There was a considerable leucocytosis, and all gave the streptococcus in smears.

My case and Loeb's were operated on early because of the apparent severity of the attack and of the type of the infection. The advanced age and the presence of chronic nephritis likewise influenced me in operating early and under local anesthesia. Even then my patient developed a blood infection, as shown by the abscesses over the malleolus and under the eye, and the second attack of erysipelas beginning on the opposite side.

The formation of these blood clots in the mastoid cells resembles the formation of the exudate in the alveoli in lobar pneumonia. The preformed cavities are filled with the bloody fibrinous exudate. On the other hand, the connection of this process with erysipelas is clearly shown in my case. The presence of bullæ in the canal and on the membrana tympani in influenzal otitis media might cause one to suspect a relationship of this condition to influenza, but the influenza bacillus was not found. The exudate also was not serous but fibrinous.

A probable point in differential diagnosis is found in that the X-ray plates in Dr. Beck's case showed very little change from the normal, while transillumination in my case gave a marked shadow. If both transillumination and X-ray examination were done on the same patient, it would probably be possible to make a diagnosis in advance of operation.

Whether one should operate early or late in these cases must be left to the judgment of the individual operator until more such cases have been reported. Ordinarily streptococcus infections require earlier operative interference than other types of infection because of the greater amount of destruction done by the streptococcus. On the other hand, the liability of the soft parts to develop erysipelas and cellulitis must be borne in mind, particularly in patients who have chronic nephritis.

XLVII.

HEMORRHAGE IN RHINOLARYNGOLOGIC WORK.*

By Arthur M. Corwin, M. D.,

Chicago.

Leaving out of account the uterus, perhaps the nose is responsible for more spontaneous loss of blood than any other region of the body, and bears its full share of accidental and operative hemorrhage as well.

And when one recalls the prime value of the blood as water carrier, food conveyor, oxygen bearer, distributer of heat, scavenger of tissue waste, equalizer of metabolic balance through the influence of hormones widely collected and widely delivered; and when one remembers the important defensive function which the blood has through its antibodies, its opsonins and its phagocytes, the rhinolaryngologist assumes added responsibility as conservator and prophylactor against blood waste.

This brief paper does not aim to summarize all the data suggested by the title. Rather shall we speak of some types of hemorrhage, emphasizing some differences among individuals of the bleeder group with which we occasionally have to deal.

It is not uncommon for physicians, even laryngologists, to apply the word bleeder or hemophiliac in a loose way to describe the episode of vicious epistaxis, or severe, possibly fatal postoperative hemorrhage marked by prolonged bleeding time and delayed coagulation, but without adequate grounds for such a diagnosis. Hemophiliac applies to a distinct type. We have in mind several cases in which the term was misapplied; for example, a boy ten years old suffered from a low grade toxemia following a mild attack of tonsillitis, which apparently cleared up locally, but left him weak and progressively losing. When we saw him, after nearly four weeks in bed, he was having a severe persistent epistaxis which oozed considerably after posteroanterior packing. He was oozing from his gums, exhibited numerous small ecchymoses on his legs and back, and subconjunctival hemorrhage. There was tenderness over a palpable spleen, and enlarged cervical glands. The viscera otherwise apparently normal. Temperature irregular, not above 102° F., at the highest. Pulse, 100 to 110. Hemoglobin, seventy-five per cent; red cells, 4,000,000; leucocytes, 9,500, practically normal. Coagulation time was much increased, the blood remaining grumous and unclotted in the basin for nearly an hour.

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The bleeding was stopped by repacking the nose with coagulose on gauze and by injection of pituitrin every six hours for three doses. On the second day he was given twenty cubic centimeters of blood serum from his brother. This, together with out-of-door treatment, arsenic and iron, was followed slowly but steadily by recovery, in spite of the fact that purpura in the course of infection is not a good sign, suggesting, as it does, fatty degeneration of the intima.

The boy had been subject to severe nose bleeds, and the family, aroused by leading questions on the part of the attending physician, had dug up a number of supposed hemorrhages among relatives. These, upon close questioning, did not prove reliable, and the possible supposition of hemorrhagic diathesis complicated by an infection dissolved into the diagnosis of purpura.

And we recognize that purpura, whether simple or hemorrhagic, is merely a symptom complex resulting from toxemia of a wide variety with no single pathologic foundation.

Rhinolaryngologic hemorrhage, purpuric in character, is an incidence in scurvy, phosphorus or snake venom poisoning, the grave cachexias, leukemia and anemia, and bacterial infections of many kinds. They do not come to nose and throat men, as a rule, except in the service of large hospitals. To differentiate these purpuric types of hemorrhage, heredity is wanting, except in rare instances; in most cases they are individual bleeders, not hemophiliacs; they frequently show various grades of blood change, and there is fatty degeneration of the capillary walls. But the blood frequently clots in normal time. In any case, the diminished clotting is nothing like as marked as in hemophilia; the blood platelets are also apt to be diminished.

The hemorrhage of venous stasis is, fortunately, more interesting to us academically than practically, for it is rather rare. The nose bleeds, and other usually trivial bleedings, subcutaneous or otherwise, which are occasionally seen in pertussis, are of this character.

The varices at the base of the tongue are rarely the site of hemorrhage, and yet their rupture may give extreme anxiety to both patient and physician unless identified. To illustrate: we recall a woman of forty years, apparently well, though when she presented herself she was a trifle short of breath, cyanotic about the lips, and suffered from a cough, with spitting of blood, which had occurred off and on frequently for some weeks.

The patient's own diagnosis was, of course, tuberculosis, but though there was well marked broken cardiac compensation, with moderate edema of the feet and ankles, etc., in the course of an old mitral lesion, the hemorrhage was not from the lungs, as was supposed, but from a break in a large vein at the base of the tongue. The treatment was naturally not local, but proper rest and cardiac medication, which was promptly effective in checking the bleeding permanently. This type of hemorrhage from throat and nose has been reported in cases of alcoholic cirrhosis.

The following is an example of laryngeal hematoma which we saw in a man of forty-five years, who had suffered from a severe subacute tracheobronchitis with severe paroxysms of cough. He was free in his use of alcohol, showed evidence of hepatic cirrhosis, with the usual gastric disturbance. After an attack of coughing, he felt a fullness in the throat, which increased to a sensation of choking, and within a short time was followed by laryngeal stridor, dyspnea and great anxiety. The pulse was rapid, and the patient sitting upright in a cold sweat, evidently in a serious condition, suggesting edema of the glottis. The examination was difficult; with the light of the house lamp we finally made out a large bluish mass obscuring the glottis entirely, but a little more to the right than to the left. With a laryngeal speculum and a long straight bistoury, wrapped with cotton except at its point, a short incision was made, followed by a burst of venous blood, giving prompt relief to dyspnea and voice. This hematoma was on the right side of the glottis, seemingly arising from the arytenoid. Incidentally it was not difficult to entirely reform the patient's alcoholic habits after that experience.

Returning to hemophilia proper, while we speak familiarly of this affection, it is not common in America, though it is said to occur twice as frequently in Germany as elsewhere. One of the very few cases the writer has ever seen was in the charity hospital at Berlin. It would be interesting to know how many cases—or rather how few—of real hemophilia have been seen by the members of the society. We venture to say they have been very few, counting the cases seen as hospital curiosities.

The speaker has seen but two cases, neither of them personal patients. In fact, we, as a profession, have not learned much more about the hemorrhagic diathesis than we were taught as students; that it is characterized by obstreperous, well-nigh uncontrollable bleeding, upon slight provocation; that it occurs spontaneously, subcutaneous and deep seated, or superficially from trivial wounds. Why it does not begin until after the second year of age, why it tends to wear out and disappear in adult years, if the patient lives long enough, is as much a puzzle as is the cause of this mysterious malady. While we were taught that it was the female who always transmitted the tendency or potentiality, and so made good Kipling's reference to her deadly reputation, we have more recently learned that she hands it invariably to a male and is herself unaffected; no female is a hemophiliac. This power of transmission through the unaffected female is also exemplified in color blindness, night blindness and diabetes insipidus, and as reported in ichthyosis and pseudomuscular atrophy. Though Virchow, and indeed earlier investigation, leads us to believe that there was in hemophilia some peculiar change in the vessel wall, a greater thinness and fragility, evidence has disproved this, or at least none has confirmed it. The three hundred and thirty-four patients collected by Grandidier revealed one hundred and sixty-nine (half of them) as epistactic cases. But the value of this ratio is minimized by the fact that Grandidier believed that females as well as males were hemophiliac in the ratio of one to four. His report contained forty-eight female bleeders, now known to be spurious in the light of the work of Bullock and Fildes, who proved that only males are actually affected. The bleeding of those forty-eight must have been due to some form of purpura or other acute condition. The real bleeder diagnosis is then summed up in:

1. Definite heredity, through unaffected females (law of Nasse).
2. Prolonged bleeding time.
3. Coagulability markedly diminished.
4. Normal blood picture except leucopenia, if anything. Blood platelets not diminished, as some have reported.
5. Origin in early youth.

We are nearly as much at sea why the hemophiliac loves to bleed as we are why normal blood loves to clot.

In view of our simple conception of coagulation, twenty-five years ago, the up-to-date biochemist seems vastly learned until we come to find that his rich vocabulary is but a cloak of ignorance, like the words idiopathic and diathesis. The foremost hypothesis concerning blood clotting may be visualized by the following verbal dominoes set on end in a row: when the first falls against its neighbor, they all go down seriatim to the last, fibrin, clotted blood. To be specific, thrombokinase or kinase, not considered present in circulatory blood, but present in shed blood, is supposedly derived from disintegration of fixed or blood cells and platelets; this acts upon another hypothetical substance, prothrombin, which in the presence of calcium salts and another substance, thrombin, somehow attracts or gives rise to fibrinogen, and out of the action and reaction springs that saving process—clotting of the blood—which Albert P. Matthews of Chicago University calls "essentially the crystallization of a protein substance, fibrin, in the form of liquid crystals which coalesce, to form fibrin strings." This is all very clear, of course. But though we know next to nothing of the how of the process, clinically the result is a vital matter.

We know physiologically that blood clotting is accelerated by raising the temperature and retarded by lowering the temperature, and yet we slap on ice bags following tonsillectomy, presumably for their reflex effect upon vasomotor constriction. But hot pledgets, not too hot, inserted directly into the wound will act as magically to check hemorrhage in the throat as in other surgical fields. Personally, we have better results by banishing the ice bag as a postoperative routine and administering five to ten grains of phenacetin with or without one-eighth of morphin for adults, after the stomach has reached postanesthetic quiet, to control early pain.

The laboratory again tells us that blood clotting is stimulated by the administration of an anesthetic, a point perhaps in favor of the good old ether anesthetic versus local anesthesia. On the other hand, says the laboratory, addition of carbon dioxid and withdrawal of oxygen lessens the tendency to clotting. This may partly account for the initial free bleeding in ordinary cyanotic gas anesthesia, or gas oxygen anesthesia, administered improperly. This is one reason why we personally do not like a socalled gas oxygen anesthetic in tonsil work, if it is given by an assistant inexperienced with it. But when administered by an expert, using an up-to-date device, accurately releasing each gas in the mixing chamber, there is no anesthetic to compare with gas oxygen in tonsil surgery. The patient thoroughly under should not show any cyanosis whatever.

Again, the physiologic laboratory demonstrates that blood received in an oil lined receptacle, under an oil film, clots slowly. This principle, applied in the use of short gauze strips boiled in vaselin at the time of operation, gives support to tissue, a clean surgical dressing, with less irritation than dry gauze or sponges. The blood does not penetrate its meshes to clot in a hard mass, difficult to remove and painful in the process. When such oiled packing is not too fine, enough seepage takes place to eliminate cocain still remaining in the tissues and to relieve congestion after the operation.

That calcium salts added to drawn blood stimulates coagulation is a familiar fact experimentally. Clinically, calcium salts have not stood up to the theory when administered internally, though we are still in the habit of giving calcium lactate or chlorid. But applied to the wound upon pledgets soaked in one or two per cent solution, as advised by Kean of Philadelphia, chlorid of calcium is of distinct benefit in checking and preventing hemorrhage. The internal use of gelatin is illogical as, according to Sahli, it does not reach the blood as gelatin. Adrenalin internally is reported to be inert by some, though advised by others, as a raiser of blood pressure; it seems to be contraindicated except for the local effect, as all of us are accustomed to use it. However, in our appreciation of adrenalin we err too often in using it too concentrated. A 1:1000 solution produces a powerful contraction and quite as radical relaxation afterward, as action and reaction are apt to correspond. In our own experienec a strength one-fourth or one-tenth the commercial solution will give better results.

But the most useful of all means by which we overcome pathologic hemorrhage is that other product of the physiologic laboratory, the knowledge that blood serum, or, under proper circumstances, whole blood, introduced into the circulation stimulates clotting as it does in drawn blood. To the report of Weil in 1907 concerning his studies of hemophiliac blood, we owe most of our brilliant results with the use of animal serum in checking hemorrhage.

In brief, we know that animal serum injected into a seemingly hopeless hemophiliac will usually save the patient. It will act as well in many cases of purpura and other little understood hemorrhagic types. Its prophylactic power when introduced into known bleeders will bring about a normal condition which in hemophilia will last for three weeks or as many months, and thereby permit surgical procedures which were otherwise impossible. Though rabbit, beef, horse and human serum produces like beneficial effect, heterologous serums are inferior to human serum in some essentials. Beef serum is not to be used because of its severe anaphylaxis. Horse serum is less anaphylactic, but at times seriously so, as illustrated in the use of diphtheria antitoxin. Blood serum from one of the same family is preferable, because of the great danger of anaphylaxis if unrelated human serum or heterologous serum is used. Fresh normal human blood serum is obtained precisely as in making a Wassermann. Serum obtained by centrifuging is less active than that obtained in the clotting process. Serum older than forty-eight hours loses in activity with age, even though it be kept properly cool and aseptic. Twenty to forty cubic centimeters, repeated in eight to twenty-four hours, is safe dosage, unless anaphylactic symptoms contraindicate; in this case human serum will fill the bill. Precipitated serum in the form of powder is approved because of its availability at critical moments. Coagulose, applied dry or in solution, has proven exceedingly reliable in epistaxis, after adenoid removal, in tonsillar hemorrhage, and in a simple obstinate case of bleeding after a tooth extraction. Coagulose is made from human blood serum, and is theoretically far superior to coagulin from horse serum. But with coagulin we have had little personal experience.

How far is the coagulation test advisable as a routine in operative rhinootolaryngology?

We have not used it invariably and have, fortunately, never yet lost a case from hemorrhage. But the same principle of nonuse applied in the field of fire insurance would be disastrous in the long run. We have in a large number of tonsil cases used ampules of pituitrin with satisfaction. M. A. Goldstein in 1913 recommended systemic coagulation tests in every case of prospective tonsillectomy, and a serum injection when the coagulation time exceeds seven minutes. This is probably good practice, but will probably never become popular in the light of majority operative freedom from hemorrhage. Fleisch emphasized the prophylactic use of animal serum in 1911. Welch reports one hundred per cent cures in the New York Lying-in Hospital in hemorrhage of the newborn, which, by the way, is not hemophilia, but more akin to purpura. On the other hand, the October 26, 1912, issue of the *Journal of the American Medical Association,* details a fatal case of hemorrhage in a child fifteen months old, from accidental wound of the tongue, despite subcutaneous use of human serum. The question is in that case whether direct transfusion of the family blood might not have resulted differently.

Surely, no laryngologist can allow a patient to die of hemorrhage without resorting to transfusion, if there is time, despite the dangers of infection, thrombosis, emboli and hemolysis. The syringe canula system of transfusion described by Dr. E. Lindermann, in the October 31st issue of the *Journal of the American Medical Association,* has made the procedure less of a bugbear. Perhaps the best recent collaboration upon the subject of serum treatment of hemorrhage is that by Dr. Henry Forbes, March, 1916, in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY. The bibliography he has gathered is well worth investigating.

XLVIII.

SUBPERIOSTEAL ABSCESS OF THE MASTOID—CURE BY PARACENTESIS.*

By Samuel Salinger, M. D.,

Chicago.

I desire to present the histories of two cases of subperiosteal abscess of the mastoid in children, with recovery through conservative treatment; also, a consideration of the anatomic features involved and a discussion of the procedure advisable in such cases.

The first is that of a boy of four years, whom I saw for the first time on May 28, 1916. He had had an acute rhinitis the week previous, which was uneventful until the appearance of pains in the right ear on May 25th, accompanied by fever lasting one day. After a twenty-four hour remission, pain and temperature reappeared, with swelling behind the auricle. Upon examination the child was flushed and restless, temperature 101° F.; pulse, 130. The right auricle was reddened and projected outward from the head, with obliteration of the posterior auricular fold, edema behind and above the auricle, redness of the overlying skin, and acute tenderness to pressure over the antrum and tip. The drum membrane was of a bluish purple color and markedly bulged, all landmarks being obliterated. A paracentesis was done, and was immediately followed by a copious discharge of pus. On the next day the discharge was less marked, the pains had increased, temperature was 101.8° F., edema had spread, and upon palpation there was distinct fluctuation. The drum was still deeply congested, and there was a slit-like perforation in the posterior superior quadrant in which pulsation was visible. I enlarged the perforation by a sweeping semicircular incision through the posterior half of the drum, inserted a drain soaked in aluminum acetate one per cent, and ordered hot wet compresses of the same solution. Within twenty-four hours the discharge again became profuse, the temperature fell to 99.4° F., and the fluctuation over the mastoid disappeared, although edema and tenderness were still marked. The treatment was persisted in as just outlined, and within four days the edema and tenderness had entirely disappeared, the discharge lessened, and the drumhead assumed a more normal aspect. Resolution was complete with intact membrane within three weeks.

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*Read before the Chicago Laryngological and Otological Society, April 17, 1917.

The second case is that of a child, aged twenty-two months, female, with no history of prior illness except that of a rhinitis of several weeks' duration. I saw her for the first time on January 17, 1917, when the mother stated that about two weeks before she noticed some whitish discharge on the baby's pillow, and a similar discharge from the left ear. There was no blood. She had not noted any discharge since that time, and the amount then was not considerable. During the past week there was a profuse nasal discharge, slightly blood tinged, and at the same time fever, restlessness and swelling over the left temporal and postauricular regions, with prominence of the auricle. Upon examination I found the temperature 102.6° F.; pulse, 140; marked edema over the left temporoparietal region, prominence of the left auricle, which was pushed forward and downward, edema and fluctuation behind the auricle. The auditory canal was not inflamed, the drum was of a delicate pink hue, the short process was easily visible, and there was no bulging. Thorough incision of the drum under anesthesia failed to reveal any pus, but was followed by a moderate flow of dark blood. A cathartic was administered and hot compresses of aluminum acetate one per cent were applied continuously over the auricle and surrounding area, including a drain into the canal. On the following morning the temperature had fallen to normal, there was a profuse discharge from the ear, the fluctuation had disappeared, and the edema was lessened. Within the next forty-eight hours the edema had entirely disappeared and the discharge had moderated. At this time the report of a smear taken on the first day of the discharge was returned, as follows: "Many pus cells, many gram negative diplococci, intra- and extra-cellular, sometimes occurring in groups. Diagnosis, gonococcus."

On the basis of this report the child had to be removed from the hospital, although a subsequent smear, examined at the same laboratory, stated merely, "gram positive cocci occurring singly and in pairs." The case, however, made an uneventful recovery at home, the discharge ceasing entirely within four weeks.

I am inclined to doubt the accuracy of the report that gonococci were found in this case, inasmuch as the subsequent smear, as well as those taken from the nose, were negative as to this germ. Also, the absence of any possible source of gonococcic infection in the family and environment of the child, as well as the fact that the micrococcus catarrhalis exhibits the same physical aspects as the gonococcus, being differentiated only through culture media and fixation tests, makes the likelihood of this case being gonorrheal remote, although not impossible. Gonorrheal infection of the middle ear was noted in three cases out of one hundred that were examined by Wingrave.[1] Two of the three occurred in infants, ten and twelve months of age, respectively, during the course of acute suppurative otitis media, and were discovered on the fourth and fifth days of the disease. Albert,[2] reporting one hundred and ten cases of acute suppurative otitis media, failed to find gonococci in a single case.

The cure of these two cases of subperiosteal abscess of the mastoid through drainage via paracentesis brings up the subject of the patency of the squamomastoid suture, which was undoubtedly the potent factor. This suture, which marks the junction of squamous with the petrous portions of the mastoid bone, is considered one of the transitory sutures of the skull, and is supposed to be closed by the end of the second year of life. Gruber[3] describes it as a line "which runs from the incisura parietalis over the mastoid to the level of the lower margin of the external auditory meatus, or even lower, and then passes forward, ending in the incisura mastoidea."

The mastoid bone, not being fully developed until puberty, presents interesting phases that have a bearing on infections during childhood.[4] [5] [33] At birth there is no distinct mastoid There is, however, a mastoid antrum, a space lying posterior to the tympanic cavity, between the petrous and the squamous bones, bounded behind by the mastoid tubercle of the former. The squamous bone sends down a postauricular process which forms the cortex of the antrum and rapidly fuses with the posterior plate of the tympanic bone, which is growing externally to form the posterior wall of the bony auditory canal. They are separated from the petrous portion of the mastoid, which grows outward and downward, by the squamomastoid fissure.[6]

During the course of the second year of life the mastoid becomes fairly distinct, and we now find it consisting of two portions, the anterosuperior or squamous portion, presenting a smooth exterior, and the posteroinferior or mastoid proper, whose surface is rough and irregular. The mastoid cells, which are formed through prolongations from the antrum, are not fully developed until puberty, although occasionally they may be found earlier. Clark[7] reports a case of complete pneumatization at three and a half years.

The patency or persistence of the squamomastoid suture has been investigated by a number of authors, whose statistics are fairly uniform. Thus, Kisselbach[8] found, in fifty-two temporal bones during the first year of life, ninety-six per cent showing the fissure more or less open. During the second year, of twenty-six bones examined, the suture was entirely or partially open in forty-six per cent, and completely closed in fifty-four per cent of the cases. His tables, which follow, show that even up to the age of nineteen the suture may be partially open, and that in a large percentage of skulls examined the suture was indicated by a series of cracks and holes in the bone.

Kirschner[9] examined three hundred skulls of all ages, and found completely open sutures in five per cent of all cases, partially or entirely open sutures on one or both sides of eighteen per cent of all cases.

Kanasugi[10] examined four thousand skulls, with the following results: Three thousand six hundred and twenty-four cases, both sutures closed; two hundred and sixty cases, both sutures partially open; in the remainder there was a partially open suture on one side, with either a completely open or completely closed suture on the other.

KISSELBACH'S TABLE OF PERSISTENCE OF THE FISSURA SQUAMOMASTOIDEA.

Fissura squamomastoidea	Entirely Open	Closed in Middle	Middle and Upper Third Closed	Middle and Lower Third Closed	Upper Third Closed	Middle Alone Closed	Lower End Open	Direction Shown Through Slits and Holes	Closed
1st Year............... (52 temp. bones)	17.3	19.23	13.46	5.77	11.54	....	28.85	....	3.85
2nd Year.............. (26 temp. bones)	....	....	19.20	3.80	....	....	23.10	....	53.90
3rd Year.............. (46 temp. bones)	4.35	....	....	....	4.35	2.18	29.13	17.39	32.60
4th Year.............. (52 temp. bones)	....	....	11.54	....	3.85	7.70	25.00	17.30	34.61
5th Year.............. (36 temp. bones)	7.98	....	....	....	5.26	2.63	23.69	13.16	47.38

6th Year............... (32 temp. bones)	....	3.13	....	....	....	6.25	6.25	3.13	81.25
7th Year.............. (30 temp. bones)	....	....	6.7	....	13.3	3.3	10.	6.7	60.
8th to 10th Year........ (24 temp. bones)	....	....	8.33	....	....	....	4.17	16.66	70.80
11th to 15th Year...... (20 temp. bones)	10.	....	10.	....	....	10.	3.3	6.7	60.
16th to 19th Year...... (18 temp. bones)	....	....	11.11	....	....	....	....	22.22	66.67

Davida,[11] on the other hand, claims he has never seen a case where the suture was entirely open, although in a series of five hundred skulls he found a more or less persistent evidence of the suture in seventy-four per cent of all bones examined up to the age of fourteen, and in thirty-nine per cent of the adults.

Aderman[12] examined five thousand one hundred and thirty-eight mastoid bones, and found a well developed suture in sixty-four. In eight hundred and forty-five cases the suture was readily recognizable, and in nine hundred and fifty-one others it was present only in traces.

An extreme case is that reported by Brühl, it being a specimen where the squamous bone was entirely separate from the rest of the temporal bone, having never fused.

The evidence thus presented indicates that in early childhood particularly we are likely to find a patent or partially open squamomastoid suture, which, leading directly into the antrum, offers a pathway for infection from the middle ear to the mastoid cortex.[14] [32] Thus the relative frequency of subperiosteal abscesses of this region occurring even in the early stages of an acute suppurative otitis media.

Mygind[15] has shown in an analysis of one hundred cases of acute subperiosteal abscess of the mastoid that seventy-eight per cent occurred during an acute suppurative otitis media, and twenty-five per cent within the first seven days.

Phillips,[30] Dench,[31] Roosa,[25] Turnbull,[26] Ayres,[27] Yamgee,[28] Zytowitsch,[24] all have reported cases of abscess of the mastoid where there were no symptoms referable to the middle ear, and where aural discharge was entirely absent. Furthermore, Brühl[16] has shown that pus may pass from the antrum through the suture without any destruction of the bone tissue.

Another important factor bearing on this subject is the remarkable resistance of the drum membrane in young children as compared to adults. The adult drum consists of very thin cuticular and mucous sheaths enclosing a delicate fibrous structure, the membrana propria. In the child the latter is more heavily developed, and is protected externally by numerous layers of epidermis. Also the inner mucous sheath is much thicker than in the adult, and presents areas thickly studded with villous formed papillæ.[16] This accounts for the cases of

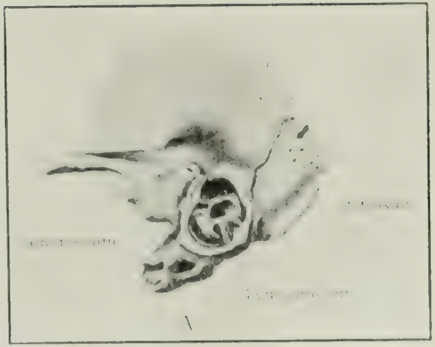

FIGURE 1.

Temporal bone of the newborn.
(Fig. 32. From Traite d'Anatomie.
Sappey.)

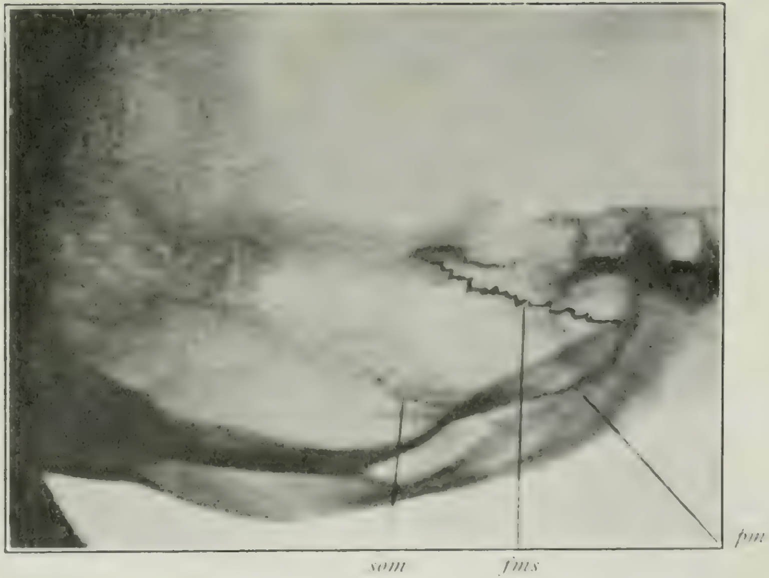

FIGURE 2.

som: Occipitomastoid suture.
fms: Squamomastoid fissure.
pm: Mastoid process.
(From Beitrage zur Pars Mastoidea. Kanasugi.)

advanced middle ear infections where the drum is unruptured presenting no pathology except for a clouding of its surface or a few injected blood vessels. The landmarks may be entirely preserved, as was witnessed in the second case above reported. While it is true that in some cases the infection may drain through the eustachian tube, this avenue is not always open. The comparatively greater width of the tube in infants, while favoring drainage, is more than counterbalanced by its more horizontal situation and the absence or incomplete development of the eustachian cartilage, the pharyngeal orifice presenting a mere slit, which is readily occluded by the slightest nasopharyngeal swelling.

These facts explain the frequent development of subperiosteal abscess of the mastoid in the absence of any marked evidence of middle ear disease, and lead up to a discussion as to the advisability of such treatment as I instituted in my cases, and the safety or danger in delaying the external operation. While the indications for doing a simple mastoid have been exhaustively discussed in various countries, and fairly uniform conclusions arrived at,[18] [19] [20] [21] [22] [23] [24] [29] the question as to the subperiosteal abscess has received less attention, doubtless because of its apparent simplicity. There can be no more universally accepted dictum than that which states the necessity for prompt evacuation of pus wherever it be diagnosed and accessible. But in the case of subperiosteal abscess of the mastoid we may have correlative circumstances wherein conservative treatment may attain the same goal with less danger to the patient than the classical or obvious measures.

We must first take into consideration the relative frequency of such abscesses in young children, the promptness with which they develop, and the anatomic reasons therefor, as indicated above. It being a fact that in over fifty per cent of all children less than two years of age the squamomastoid sutures are more or less patent, is it not reasonable to expect that drainage in the direction of the original focus may be made as effective as the progress of the pus in the opposite directions?[17] In other words, if the squamomastoid suture can transmit pus from the antrum to the cortical periosteum, it can also transmit pus from the mastoid cortex through the antrum into the middle ear. However, the conditions under which this retrograde drainage may be attempted are not to be found in all cases, and, therefore, the indications for such measures are limited. It is only in an acute case, where the drum has not been perforated and the middle ear drained, or where the perforation is inadequate, that one may counsel conservatism, and then only with the proviso that there be no other threatening symptoms that would indicate necrosis in the mastoid adjoining other vital structures, such as the lateral sinus, the dura of the middle fossa, or the labyrinth. Furthermore, where drainage through the tympanic membrane has been effected, and the fluctuation and edema of the mastoid fail to promptly disappear, there can be no question as to the necessity of immediate incision into the bone.

In spite of the fact that even spontaneous resolution in mastoiditis has been considered possible, as has been attested to by Politzer,[18] I am inclined to the view of Mygind,[29] who states that a true osteitis of the mastoid cannot heal spontaneously without leaving residues that will be heard from later. However, the cases such as I have described do not come under this heading. Subperiosteal abscess can and does develop without perforation of the mastoid cortex or accompanying osteitis in the manner above described, and it is this type of abscess that will develop in the very earliest days of the otitis media in such cases as have a persistent squamomastoid suture. It is in these cases particularly, where drainage through the middle ear has not been established, and in the absence of any threatening symptoms, that the classic Wilde incision may with safety be deferred until thorough paracentesis and antiphlogistic measures have been instituted.

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XLIX.

FOREIGN BODIES IN THE ESOPHAGUS—AN ANALYSIS OF FIFTY CASES.*

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The symptoms caused by the presence of a foreign body in the esophagus vary to a great degree. Inasmuch as the accident occurs most frequently in young children, the difficulty of eliciting a definite history is obvious. As is to be expected, the most prominent symptom is the difficulty in swallowing. This may be so slight as to almost pass unnoticed, or, conversely, so marked that even fluids will not pass through the esophagus. In some patients, after the first irritation has subsided, the symptoms almost entirely abate, and doubt may arise as to whether or not a foreign body is present.

In other instances, symptoms persist after the foreign body has been removed or has passed into the stomach. In young children, if the obstruction is at all marked, there is a continual drooling of saliva, with an excess of secretion in the mouth. The child may hold its hand to the neck or incline the head to one side. Shortly after swallowing there is regurgitation of food. In taking the history this is usually improperly called vomiting. In patients in whom an indirect examination can be made, the pyriform fossæ are seen to be filled with secretion. When the esophageal obstruction is marked, this usually amounts to a considerable quantity, and by trickling into the larynx and trachea may excite cough. Large foreign bodies by their pressure on the tracheoesophageal wall produce respiratory disturbances to such an extent as to lead to the suspicion that the foreign body is in the air passages. Pain is intense, according to the nature of the foreign body and the local reaction which it has brought about. It may be referred to the seat of the foreign body, to the posterior cervical region, if the latter is high up, or along the vertebral column, if it is in the region of the hiatus. It may be present constantly or only on attempted deglutition. From its location and its interference with swallowing, it is difficult to tell whether the foreign body is located in the upper portion of the esophagus or the pyriform sinus.

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The systemic effect of the presence of foreign bodies depends upon their nature, the time they have been present, the amount of obstruction they are causing, and the local changes or reaction they have produced. A careful observation of the temperature will usually show a slight elevation, even in those cases in which the symptoms are few or indefinite The complications that may ensue from their presence are esophagitis, periesophageal abscess, mediastinitis, or ulceration and perforation of the tracheoesophageal wall. The long continued presence of a foreign body will lead eventuallv to contractures and stricture of the esophagus.

One of the most important aids in diagnosis is the roentgenograph. For large foreign bodies the single picture is satisfactory. For smaller ones, when there is a doubt as to whether the foreign body is in the esophagus or trachea, stereoscopic plates are of greater value. The angle at which pictures are taken is important. I have seen pictures of the same patient made by different roentgenologists in which there was a variation of at least an inch in the apparent location of the foreign body. one plate showing it lower than it actually was. The necessity for having the clothing completely removed from the field cannot be too strongly emphasized. Otherwise, one may spend considerable time looking for an imaginary foreign body. The absence of a shadow does not eliminate the presence of a foreign body. In adults, fish or meat bones high up in the esophagus are almost impossible to interpret on a plate, on account of the impossibility of eliminating areas of ossification in the laryngeal cartilages. Certain types of large buttons, reputed to be made of vegetable ivory, do not give a shadow. The following description will illustrate the above point:

A young child was said to have swallowed a large button. There was considerable difficulty in swallowing. The roentgenogram showed a fair sized button in the region of the hiatus. Esophagoscopy failed to reveal the foreign body. Immediately following the examination another roentgenogram showed that the button had disappeared. Although not quite satisfied that the trouble was over, I allowed the child to go home with instructions as to the necessary observation. The original symptoms persisted, and a few days later the child was returned to the hospital. The second examination showed the button in the usual location, whence it was easily removed. It was of vegetable ivory and did not give a shadow. The button that was visible in the plate, and which I had looked for the first time, was of bone and was attached to the waist.

The question of anesthesia is an important one. While there are not the same contraindications to the use of general anesthesia for work in the esophagus as in the trachea or bronchi, it may be said that this is only occasionally necessary. Cocain may be used quite satisfactorily in adults, but should not be employed in children. In operating the patient should be in the recumbent position. It is well to have the table arranged so that the head can be lowered quickly in case the forceps slips off the foreign body. By doing this we are able to have the benefit of the influence of gravity, the foreign body remaining in the esophagus and not passing into the stomach. Foreign bodies that have lodged in the upper portion of the esophagus may easily traverse the remaining part, if they have been loosened.

The problem connected with the extraction of foreign bodies is almost entirely a mechanical one. It is well to have as definite an idea as possible as to the size and composition of the object that has been swallowed. This may usually be obtained from the patient or parents, or from a careful study of the roentgenogram. A comparison with an object of similar nature is advisable. This was of much value in one patient with a jackstone in the esophagus. Much difficulty was encountered by reason of the forceps repeatedly slipping off one of the prongs. By questioning the mother of the patient it was found that the ends of some of the prongs were smooth while the others were knobbed. By turning the jackstone a knobbed end was seized and the foreign body brought up to the lower border of the cricoid cartilage, where it held. It was impossible to bring it out without using enough force to bring the cricoid with it. By keeping a firm hold with the forceps and introducing my finger into the mouth of the esophagus, the jackstone was rotated and removed.

Several years later, an infant with a similar foreign body came under my care, and I anticipated having the same difficulty. However, on seizing the foreign body, it came out so easily that I lost it in the mouth. On recovering and examining it, I found that several of the prongs had been greatly worn down and that none of them was knobbed.

In the case of sharp pointed objects, such as common or safety pins, irregular or jagged pieces of bone or metal, extreme caution must be used in order not to perforate or tear the esophagus. One must resist the temptation to seize and remove the foreign body as soon as found, regardless of what damage may be done. It is much better to work slowly and carefully, planning definitely the procedure to be followed, even though another examination may be necessary. When traumatism has resulted from ill advised attempts at removal, the operation is made much more difficult. Inflammatory swelling, edema, transfixion or impaction of the foreign body also add to the difficulties.

The danger attached to the removal of foreign bodies by any other means than under direct vision cannot be too strongly emphasized. The use of probangs or umbrella bougies is rarely justified. With pointed, rough, or jagged obstacles the possibilities for harm are infinite. Even with smooth articles there is considerable danger, as is illustrated by the following instance:

A very young child was admitted to my service at the County Hospital, with a history of having gotten a piece of pig's knuckle in the throat. A physician who was called was unable to remove it, and under the theory of what would not come up must go down, he pushed it, as he thought, into the esophagus. The result was the foreign body went through the esophageal wall, infection was set up and the child was in a serious condition at the time of admission. The bone could be felt in the tissues of the neck and was removed by an external incision. Death resulted from the infection which was present.

I have outlined below an analysis of some fifty cases of foreign body in the esophagus. The following table gives the age of the various patients:

7 months to 1 year	5
1 to 2 years	13
2 to 3 years	6
3 to 4 years	9
4 to 5 years	2
5 to 10 years	4
10 to 15 years	2
15 to 20 years	1
20 to 30 years	2
30 to 40 years	2
40 to 60 years	3
63 years	1
Total	50

The character of the foreign bodies was as follows:

Coins or disc shaped objects	29
Bone	6
Pins	6
Metallic substances	6
Animal matter	2
Marble	1
Total	50

Location of foreign bodies:

Below plica cricopharyngeus	40
Hiatus	4
Above plica	4
Middle third esophagus	2
Total	50

Results:

Dislodged and passed into stomach	2
Partial removal	1
Extraction	47
Total	50

The problems connected with this series of foreign body cases briefly were, first, those with points, in which it was necessary to avoid injuring or perforating the esophageal wall; secondly, those in which there was considerable reaction with impaction of the foreign body; third, one in which the foreign body could not be brought out except by the aid of the finger, on account of its size; and lastly, one in which a stricture had to be dilated before the foreign body could be found. One patient, a child, had two operations, six months apart, for the same kind of a foreign body. Another patient, a hysterical girl, with a pin swallowing habit, was examined four or five times, and altogether about seven pins were removed from the pharynx and hypopharynx. Radiographs showed the presence or absence of pins with such monotonous regularity that our suspicions were at last aroused, and the girl confessed that her accidents were entirely voluntary. The only fatal case was in a child with a coin in the esophagus. There had been considerable traumatism, and death resulted from infection following a tracheoesophageal fistula.

FIFTY CASES OF FOREIGN BODY IN THE ESOPHAGUS

Age	Foreign Body	Location	Anesthetic	Problem	Point of Seizure	Result	Remarks
10 months	Ring	Hooked over arytenoid and projecting into mouth of esophagus	Ether		Presenting edge	Extraction; cure	Symptoms absent.
2 years	Belgian coin	Below plica cricopharyngeus, five days	Ether	Unable to locate	Presenting edge	Extraction; death	Marked traumatism. Death from tracheoesophageal fistula.
5 years	Marble	At hiatus, five days	Ether	Difficulty in grasping		Extraction; cure	
1 year	Penny	Below plica cricopharyngeus, one day	Ether		Presenting edge	Extraction; cure	
17 months	Penny	Below plica cricopharyngeus, four days	None		Presenting edge	Extraction; cure	

2 years	Nickel	Below plica cricopharyngeus	None		Presenting edge	Extraction; cure	
13 months	Penny	Below plica cricopharyngeus	Ether		Presenting edge	Extraction; cure	
5 years	Whistle	Below plica cricopharyngeus, four days	Ether		Presenting edge	Extraction; cure	
30 years	Meat bone	In right pyriform sinus, one day	Ether, Cocain	Marked edema right half of hypopharynx	Presenting part	Extraction; cure	Overlooked, on account of edema at first examination.
14 months	Metal toy	Below plica cricopharyngeus, several days	Ether			Dislodged; passed two days later	
17 years	Meat bone.	Below plica cricopharyngeus, one day	Ether		Presenting edge	Extraction; cure	
3 years	Brass button	Below plica cricopharyngeus, two days	None		Presenting edge	Extraction; cure	

Age	Foreign Body	Location	Anesthetic	Problem	Point of Seizure	Result	Remarks
18 months	Part of watch movement	Below plica cricopharyngeus, one and one-half days	Ether	To avoid tearing wall on account of projecting pinions	At pinion	Extraction; cure	
8½ months	Open safety pin	Below plica cricopharyngeus, four days	Ether	Point up		Extraction; cure	Pin closed with Arrowsmith forceps.
3 years	Nickel, two pennies	Below plica cricopharyngeus, three days	Ether		Presenting edge	Extraction; cure	
2 years	Telephone slug	Below plica cricopharyngeus, seven days	Ether		Presenting edge	Extraction; cure	
20 months	Quarter	Below plica cricopharyngeus, several days	Ether		Presenting edge	Extraction; cure	
19 months	Button	Below plica cricopharyngeus, six days	Ether		Presenting edge	Extraction; cure	

3 years	Telephone slug......	Below plica cricopharyngeus, two and one-half days...........	None......	....	Presenting edge........	Extraction; cure........	
19 months	Telephone slug......	Below plica cricopharyngeus, five days....	None......	....	Presenting edge........	Extraction; cure........	
2 years	Button....	Below plica cricopharyngeus, thirteen days	None......	....	Presenting edge........	Extraction; cure........	
7 months	Open safety pin....	Below plica cricopharyngeus, several days .	None......	Point up.....	....	Extraction; cure........	Pin closed with Arrowsmith forceps.
48 years	Chicken bone.....	Hiatus, three days....	Cocain....	Transfixed. Rotation...	Grasped near end and withdrawn...	Extraction; cure........	
3 years	Two pennies......	Below plica cricopharyngeus, thirteen days	None......	....	Presenting edge........	Extraction; cure........	
16 months	Metal toy goat......	Below plica cricopharyngeus, three days...	None......	Rotation.....	Head........	Extraction; cure........	

Age	Foreign Body	Location	Anesthetic	Problem	Point of Seizure	Result	Remarks
48 years	Part of fish fin.......	Above bronchial crossing, one day.........	Cocain....	Impaction...	Presenting edge........	Extraction; cure........	
26 years	Picture button....	Below plica cricopharyngeus, three hours..	Cocain....	Pin portion partly open. Rotation ...	Presenting edge........	Extraction; cure........	
2 years	Button....	Below plica cricopharyngeus, four and one-half months.........	None......	....	Presenting edge........	Extraction; cure........	Symptoms respiratory. Diffuse bronchitis.
7 months	Open safety pin....	Middle third of esophagus, seven days......	None......	Point down closed by drawing into tube........	Ring portion.	Extraction; cure........	
4 years	Iron washer........	Middle third of esophagus., a number of months.............	Ether.....	Dilatation of stricture before foreign body could be found......	Presenting edge........	Extraction	

3 years	Jackstone	Below plica cricopharyngeus, four hours...	None.....	Rotation to grasp knobbed prong...	At knobbed prong......	Extraction; cure........	Could not be pulled above cricoid. Held with forceps and delivered by rotating with finger.
11 years	Pins.......	In hypopharynx......	Cocain....	Hysteria.....	....	Extraction; cure........	Several operations performed.
6 years	Nickel.....	Below plica cricopharyngeus.............	None......	....	Presenting edge........	Extraction; cure........	
2½ years	Penny.....	Below plica cricopharungeus, two weeks...	None......	....	Presenting edge........	Extraction; cure........	
17 months	Collar button, hinged	Below plica cricopharyngeus, 10 hours.....	None......	....	Presenting edge........	Extraction; cure........	
14 months	Tin button	Below plica cricopharyngeus, four weeks...	None......	....	Presenting edge........	Extraction; cure........	
3 years	Penny.....	Below plica cricopharyngeus, four days....	None......	....	Presenting edge........	Extraction; cure........	

Age	Foreign Body	Location	Anesthetic	Problem	Point of Seizure	Result	Remarks
13 months	Jackstone	Below plica cricopharyngeus, seven days	None		At prong	Extraction; cure	
41 years	Meat bone	Below plica cricopharyngeus, two days	Cocain	Impacted, rotation on account of sharp prolongation		Extraction; cure	
27 years	Half dollar	Hiatus, thirty-two hrs.	Cocain	Impaction	Presenting edge	Extraction; cure	
7 years	Nickel	Below plica cricopharyngeus, one day	None		Presenting edge	Extraction; cure	
16 months	Meat button	Below plica cricopharyngeus, two days	None			Extraction; meat button dislodged, passed later.	
10 months	Open safety pin	Below plica cricopharyngeus	None	Point up	Grasped at point and withdrawn	Extraction; cure	

35 years	Bone	Hiatus	Cocain	Transfixed	Grasped and drawn upward. Slipped off forceps	Dislodged, passed into stomach	
4 years	Quarter	Below plica cricopharyngeus, two and one-half days	None	Impacted	Presenting edge	Extraction; cure	
3½ years	Nickel	Below plica cricopharyngeus, one day	None		Presenting edge	Extraction; cure	
62 years	Meat	Below plica cricopharyngeus, three days	Cocain	Impacted, friable		Extraction; cure	
12 years	Pin	Above plica, two days.	Cocain	Point in tissues	At head	Extraction; cure	
3½ years	Penny	Below plica cricopharyngeus, two and one-half days	None		Presenting edge	Extraction; cure	Six months before had similar accident.
3 years	Pearl button	Below plica cricopharyngeus, six hours	None		Presenting edge	Extraction; cure	

L.

REPORT OF A CASE OF NASAL SARCOMA CURED BY RADIUM.*

By Otto J. Stein, M. D.,

Chicago.

F. L., aged twenty years, presented himself July 2, 1916, complaining of complete nasal obstruction, profuse attacks of epistaxis and severe headaches.

Examination revealed the following: A young man, tall, dark complexion, anemic, poorly nourished, mouth breather, right eye protruding so that the lids could not cover the ball, anterior nares excoriated from nasal discharge, nasal bridge widened and tissues round about thickened, altogether giving the frog-like facies. Right nostril was entirely blocked by a pale colored, fairly hard mass, excepting in spots where there was local necrosis showing areas of sloughing surrounded by hemorrhagic oozing. The tissue bled freely on manipulation, and especially so on application of adrenalin. As far as could be determined, the tumor seemed to originate from the right ethmoid region. The septum was intact and with no adhesions between it and the tumor. The left nostril was not invaded, being open. The nasopharynx was entirely filled by the tumor plugging both posterior nares. A probe introduced into the left or open nostril could be passed around the mass in the epipharynx. Bleeding always resulted from such instrumentation. The patient suffered acutely from constant headache and the attacks of epistaxis. The right sinuses showed dark on transillumination. The skiagraph demonstrated shadows in the region of the right antrum, ethmoid and nostril.

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*Read before the Chicago Laryngological and Otological Society, April 17, 1917.

Examination of the eye by Dr. George Suker revealed optic nerve atrophy, no swelling of discs, veins some tortuous, arteries likewise, disc edges blurred and adjacent retinal area shows grayish cast. Has a large and painful mass of cervical lymph gland on left side of neck; scar shows incision of former years. No family history of malignancy, no cough, no other evidences of pulmonary tuberculosis. Wassermann negative. Nasal obstruction came on gradually some months before presenting himself. A diagnosis of sarcoma was made from the patient's age, the history, general appearance and local findings. This was confirmed by microscopic examination of tissue removed, which showed small round cell sarcoma.

On our refusal to operate the patient disappeared. Presenting himself at the County Hospital, an attempt was made to remove the tumor by way of the anterior nares. The hemorrhage was severe. An infection resulted, necessitating an external incision over right malar region to liberate pus. Sections of the tumor removed demonstrated round cell sarcoma. After six weeks the patient again presented himself to us for relief. His appearance was similar to what it was when first seen, only his condition was worse. He did not look as if he could live much longer, being very weak and anemic, and on account of pain in his head had little rest day or night excepting what he got from sedatives. He was immediately placed on radium treatment. One hundred milligrams of radium was applied uninterruptedly for five hours on three consecutive days. Nineteen days later this was repeated, excepting that it was continued for six hours each day. Thirty days later fifty milligrams was applied for four hours, and sixty days later fifty milligrams was applied on two consecutive days for six hours each. In addition, radium was applied to the large painful gland mass on left side of neck. A total of forty-one hundred milligram hours of radium was used about the nose and twenty-one hundred and twenty-five milligram hours of radium was applied to the enlarged glands. The details of the radium treatment is appended, kindly compiled for me by Dr. Albert Woefel of the Physicians' Radium Association, who furnished the radium used.

RADIUM TREATMENTS OF CASE OF SARCOMA OF NOSE

Dates	Dose	Screen	Exposure	Position	Mgm.Hrs.
Sept. 25, 26, 27.	50 mg.	1 mm., lead-black paper and rubber.	5 hours. .	Rt. post. nares. .	750
Oct. 16, 17, 18. .	50 mg	1 mm. plat. and rubber	6 hours each. . . .	Rt. post. nares. .	900
Nov. 21.	50.	1 mm. plat. and rubber	4 hours. .	Rt. post. nares. .	200
Jan. 23, 25.	50.	1 mm. plat. and rubber	6 hours each. . . .	Rt. post. nares. .	600
					2450
Sept. 25, 26, 27. .	25 and 25. .	2 mm. lead and rubber.	5 hours each. . . .	Varying positions around orbit.	750
Oct. 16, 17, 18. .	25 and 25. . .	2 mm. lead and rubber.	6 hours each. . . .	In V-shaped position on temporal side of right orbit. . . .	900
					1650

Nov. 14	50 and 50 and 25 mg.	2 mm. lead and rubber	6 hours.	On left cervical glands	750
Nov. 16	50 mg.	2 mm. lead and rubber.	10 hours.	On left cervical glands	500
Nov. 17	50 and 25 mg.	2 mm. lead and rubber.	6 hours.	On left cervical glands	450
					1700
Jan. 23	25 mg. (flat)	1.5 mm. lead and rubber	4½ hours	On upper part of neck	112
Jan. 25	25 mg. (flat)	1.5 mm. lead and rubber	6½ hours	On upper part of neck	163
Jan. 27	25 mg. (flat)	1.5 mm. lead and rubber	6 hours.	On neck	150
					425

In employing the remedy the object was to use large doses over a medium period of time, correctly screened and properly applied within the nose and on the face to secure the greatest benefit from cross firing. The immediate results from this treatment were brilliant. All of the severe pain and the nasal hemorrhage disappeared before a week went by. A week later he was breathing through his left nostril, and some air could be detected passing through on the tumor side or right nostril. Three months later respiration through both nostrils was normal, and he could blow nostrils naturally. Inspection showed the progressive shrinking of the tumor and disappearance of all ulcerations. The rhinoscope demonstrated the tumor springing from the posterior ethmoid region. This would explain the appearance and changes about the right eye —namely, exophthalmus, nerve atrophy, tortuous veins, etc. The optic nerve and vessels are more vulnerable in the neighborhood of the sphenoid sinuses; but where a posterior ethmoid cell occupies this relative position, then the nerve becomes correspondingly more susceptible from this source. From various anatomic studies this relationship of the posterior ethmoid cell to the nerve occurs in a small per cent of cases. Meckel's ganglion, the source of sensory impressions in and about the nose, orbit and sinuses, nestles in the sphenomaxillary fossæ, just below the posterior ethmoid cell and in front of the sphenoid and back of the apex of the antrum of Highmore. A tumor in this region can produce severe pain corresponding to that complained of by the patient.

On last examination of the patient no evidence of tumor could be seen within the nose or nasopharynx, although I believed some still existed within the ethmoid labyrinth. Three weeks after seing him last he was reported as being confined to bed at home with a temperature, pain in abdomen, nausea, constipation, tympanitis, and a cutaneous eruption over left half of face. Not being able to visit him, he was directed to be sent to the hospital for examination. Disregarding this request, he remained at home, under the care of his family physician, who reported to me a few days later that the patient was suffering from metastasis and that the malignancy was manifesting itself in the intestines, causing severe pain, tympanitis, vomiting, and great weakness. It was stated that he would not survive this serious sequela. It was further stated that, aside from a cutaneous eruption on the left side of face, the patient had no symptoms whatsoever referable to his head.

After ten days of illness he died. A request for an autopsy was made and granted. I opened and examined the head. Dr. McKerrihan of the Postgraduate Hospital examined the abdomen. After removing the calvarium and brain, exposing the interior of the base of the skull, examination showed no invasion. The entire tegmen of the right orbit, frontal sinuses and nasal fossæ was removed, exposing the interior of these respective cavities, which showed neither the presence of or any evidences of their invasion. The antrum and sphenoid were likewise free. The posterior ethmoid cavity was large, extending far backward, and overlying in part the neighboring sphenoid cell, which was very small. The lining membrane appeared greatly hypertrophied, the optic nerve course was alongside the cell. The middle turbinate was not present excepting a small bit posteriorly. The anterior ethmoid cells had been broken down by previous operation. No tumor could be found anywhere. These findings affirm the previous statement that the tumor had its orgin in the right ethmoid region, and owing to the anatomic arrangement present, the nerves and vessels were subject to pressure. As far as the head was concerned, the patient was cured of his sarcoma.

The examination of the contents of the abdominal cavity disclosed, aside from a distended gall bladder, a perforated appendix lying in a bed of pus and exudate. Neither within nor upon the surface of the body was found evidence of metastasis. The eruption on the face was herpetic, toxic in origin, and an early manifestation of an infection that was overlooked. The patient most unfortunately came to an untimely death as the result of peritonitis caused by perforating appendicitis.

LI.

PERISINUS ABSCESS WITH APPARENTLY NORMAL SINUS, FOLLOWED BY EXTENSIVE THROMBOSIS WITH MULTIPLE METASTATIC ABSCESSES. REMOVAL OF CLOT FROM SINUS AND BULBUS. JUGULECTOMY—RECOVERY.

By Otto Glogau, M. D.,

New York City.

On February 27, 1916, I was called by Dr. H. Fried to see H. N., sixteen years of age. On February 19th the patient had been taken sick with la grippe. The following day, without much preceding pain, the left ear started to discharge. Two days afterwards severe pains over the mastoid process set in and continued to the date of examination. The ear had been discharging freely, the temperature being around 102°.

Local inspection revealed a perforation in the posterior lower quadrant of the drum membrane with free drainage. The posterior upper canal wall was slightly sagging. Severe pressure pains over the tip of the mastoid process and over the antrum region were present. The upper and posterior aspect of the mastoid bone were not sensitive. Temperature, 103°. The patient was advised to go to the hospital for observation and probably operation. I did not hear from the patient till March 2d, when I was called again to see him. The sensitiveness had extended over the entire mastoid bone, being especially severe at the tip and the posterior border. The canal wall was bulging forward to a marked degree, and there was free and copious discharge from the ear. Temperature, 103°. No redness or swelling behind the ear. The patient was then removed to the Philanthropine Hospital and immediately operated upon.

After the usual semilunar incision and retraction of the periosteum, the mastoid bone was exposed and showed a livid discoloration. After chiseling open the very thin cortex, the entire mastoid cavity proved to be destroyed and filled out with necrotic granulation tissue and pus. There was a perisinus abscess which had destroyed the bony wall of the sigmoid sinus to the extent of two centimeters. The sinus wall, both as to color and touch, appeared normal; pulsation was clearly noticeable. The antrum was found to be necrotic, but no probe could be introduced into the aditus. The middle ear had apparently been separated from the mastoid process by proliferation around the antrum. The antrum was curetted and a communication established with the middle ear. The upper part of the wound was closed by skin and periosteal sutures, the wound cavity filled with gauze strips. The pus from the mastoid wound contained streptococci.

The following day the temperature rose to above 105° and dropped within a few hours to 102°. During the next few days one or more such fluctuations occurred during twenty-four hours. On the third day after the operation the rise of the temperature to 105.6° was preceded by a chill and followed by exhaustive sweating. A blood culture was taken and no streptococcemia was found. The pulse rate varied between seventy-four and one hundred and twenty-eight, but was mostly around one hundred. There were severe pains on the affected side of the head; mostly referred to the mastoid region. The diagnosis of thrombosis of the sigmoid sinus was made, and immediate removal of the thrombus advised. Dr. S. Oppenheimer, in consultation, agreed with the diagnosis, and also advised immediate operation.

The operation was performed on March 10, 1917. After thorough sterilization of the head and neck, the mastoid wound was reopened.

The sigmoid sinus was exposed to the extent of three centimeters. The sinus was found to be inflamed, of dark grayish color, and its wall very much thickened. No pulsation could be felt. A piece of gauze was pressed by the assistant against each end of the exposed sinus, while its wall was slit open by means of a curved and pointed bistoury. No bleeding took place from either portion of the sinus. The sinus was filled out by a rather soft clot of dark grayish color.

In order to avoid the dislodging of particles of the clot into the general circulation, I decided to ligate the jugular vein prior to curetting the sinus. The mastoid wound was temporarily closed with iodoform gauze. The incision in the neck was begun at a point of the anterior margin of the sternocleidomastoid muscle, horizontally posterior to the angle of the jaw, and continued downward parallel to the margin of the muscle for two inches. The jugular vein was exposed in the regular way, after opening the common sheath of the vessels. The vein was found to be a solid mass of the diameter of a small finger, of dark grayish color. As the thrombosed portion seemed to extend further down, the vein was exposed to the clavicle and found to be thrombosed to that distance. The internal jugular vein was then separated from the pneumogastric nerve and the carotid artery by means of a dull dissector, and a double catgut ligature was passed above the entrance of the facial vein and another one at the lower end of the thrombosed portion of the vein between the two sets of ligatures excised. A number of cervical glands, enlarged, inflamed and apparently infected, were also removed. I pass around the specimen of the excised portion of the thrombosed vein.

The gauze from the mastoid wound was then removed and the incision prolonged to meet the one on the neck. The sinus was then curetted in the direction of the bulb. Masses of decaying clot tissues were thus removed, but no bleeding occurred. More and more bone tissue had to be removed in the direction of the jugular fossa, and still no bleeding occurred upon removing the clot by means of the curette. I then proceeded, according to the method of Grunert, with dull dissection into the depth, till the external bony margin of the jugular foramen was reached. The bony bridge between the latter and the previously exposed lowermost end of the sigmoid sinus was then carefully chiseled away. The bulb of the vein became thus accessible to the knife, and the incision from the lower end of the sigmoid sinus was thus prolonged over the bulb into the internal jugular vein. After the removal of all thrombosed masses the formerly closed vascular system consisted of a groove open to the outside with its inner wall consisting of the slit open wall of the sigmoid sinus, bulb and internal jugular vein. In order to expose the torcular end of the sigmoid sinus, an incision was made vertically to the semilunar over the mastoid, and the tissues retracted. The bone had to be removed for one more inch backward before the curetting of the sinus resulted in free bleeding from its torcular end. The entire wound was then flushed with normal saline solution, the edges of the sinus wall pressed inward by gauze strips, the mastoid cavity filled with gauze packing and closed at its upper portion. A cigarette drain was inserted into the jugular wound in its entire length and the incision closed, the drain protruding near the clavicular and the mastoid end of the incision. The temperature after the operation remained septic for over a week; the temperature, however, would daily rise less high and gradually drop lower.

On the fifth day after the operation a metastatic abscess, in the region of the buttocks, on the left side, was noticeable. The next day a few ounces of pus were evacuated, the cavity was curetted and packed with iodoform gauze. Two days afterwards another metastatic abscess was detected at the occipital region of the scalp, and treated in a similar way. Two days later a swelling around the thyroid portion of the larynx was noticed, which soon started to fluctuate. Upon incision two ounces of pus were evacuated. The patient had suffered from choking sensations, lack of air, and pains in swallowing, which were relieved by the evacuation of the pus. While the other two metastatic abscesses healed within a short time, the metastatic abscess around the larynx suppurated for four more weeks. Due to the excision of the internal jugular vein, the left side of the neck and face was swollen for a short time. The mastoid and jugular wound were frequently dressed. The patient made an uneventful recovery, and the external wounds closed entirely within two months after the operation. Subsequent bacteriologic examinations of the blood, during the formation of the metastatic abscesses, at no time revealed streptococcemia.

The case offers the following interesting features:

Perisinus abscess with apparently normal sinus wall at the primary mastoid operaion. Septic fever curve, characteristic of sinus thrombosis, with negative blood cultures, although the pus from the mastoid shows pure streptococci. Thrombosis of entire internal jugular vein and inflammatory hypertrophy of the cervical lymph glands, without any changes around the neck pointing to this condition.

The thrombosis of the jugular vein was practically only detected while its ligation, prior to the curetting of the thrombosed sinus, was undertaken. The question whether or not the jugular vein shall be ligated before curetting a thrombosed sinus has not been settled as yet. The findings of this case are, however, very suggestive of the exposure and inspection of the jugular vein, even in those cases where the appearance of the thrombus within the sigmoid sinus does not imperatively demand the ligation of the vein.

LII.

SUSPENSION LARYNGOSCOPY.*

By H. L. Pollock, M. D.,

Chicago.

When one presents a paper before a society like ours there are certain conditions which are to be met. They consist in (1) presenting something entirely new or original, either in subject matter, technic or instruments; (2) an improvement or modification of something already presented; (3) a résumé with conclusions drawn therefrom, and (4) an emphasis of a well known condition by presenting it from time to time and adding something new, either favorable or unfavorable, to that which is already established.

My object in discussing suspension laryngoscopy is not to add anything materially to the technic, nor have I many new instruments or appliances, but simply to call your attention to the fact that this method has been very much neglected in the hands of the otolaryngologist through this country. When one considers the great advantages, either diagnostic, medical or surgical, this method of doing intralaryngeal work has over the indirect method, it is scarcely conceivable that so few laryngologists are familiar with the technic which is so easily performed with such simple apparatus and instruments. That this is true is evidenced by the large number of visitors at our clinic at the North Chicago Hospital, who for the first time have witnessed suspension laryngoscopy.

In 1915 my associate, Dr. Joseph C. Beck, read a paper before this society, giving our experience in over two hundred cases where suspension had been employed. Since that time we have had many more interesting cases, and have met some new conditions. The histories and course of some of these I will give later.

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*Read before the Chicago Laryngological and Otological Society, May 22, 1917.

At that time he cited several conditions in which suspension was impossible, but since then Dr. Lynch and others have greatly improved the technic, and some of these objections and difficulties have been overcome. In the beginning, excepting a few cases of anatomic or pathologic changes, such as a tumor at the base of the tongue or the epiglottis, ankylosis of the jaws, etc., our greatest difficulty was in those cases where there were no teeth in the upper jaw, or only a few fangs, or where the teeth had been loosened by pyorrhea. Since employing Lynch's modeling compound aluminum plate, this difficulty has to a greater extent been done away with.

The advantages which this method possesses over the indirect, except in the routine office practice, in diagnosis and treatment is so marked that it would be a waste of time to begin to enumerate them, and the disadvantages are comparatively so few that they are scarcely worth mentioning. In adults, for a routine superficial laryngeal examination, the indirect method usually answers the purpose, but we have had cases where, notwithstanding perfect cocainization, an examination was impossible. In children and infants, however, direct examination is impossible, except under general anesthesia. To be sure, suspension laryngoscopy is quite a formidable procedure, if we consider the time required and the amount of cocain employed, but the results in those where it is indicated justifies our endeavors, and one feels fully recompensed for the time spent by the favorable results obtained.

The indications for the use of suspension are about the same as those described in Dr. Joseph C. Beck's paper, which are as follows:

1. Carcinoma.
2. Adenocarcinoma.
3. Papilloma.
4. Fibroma.
5. Angioma.
6. Syphilis.
7. Tuberculosis.
8. Rhinoscleroma.
9. Cicatricial stenosis.

10. Paralysis.
11. Foreign bodies.
12. Burns.
13. Asthma.
14. Posttraumatic tracheal stenosis.
15. Bronchiectasis.
16. Suction of secretion for bacteriologic examination.
17. Direct intubation.
18. Simple laryngitis.
19. Posttyphoidal chondritis.
20. Atrophic laryngotracheitis.
21. Hysteric aphonia.
22. Suspected thymus enlargement.
23. Tracheal compression by thyroid enlargement.
24. Aortic aneurism.
25. Normal cases.

During the past eighteen months we have not had a single case in which suspension, either under local or general anesthesia, could not be successfully performed. There was one case, however, which I will mention later, in which suspension was very easy but the tumor could not be reached; the reason therefor will also be explained.

As stated before, general anesthesia is necessary in children and infants, and this is given either by the blowing vapor method through the mouth or through the tracheotomy tube, if such was previously necessary. In practically all the adults local anesthesia sufficed, but just recently we had one case where, notwithstanding a perfect local anesthesia, suspension could not be performed, and a general anesthesia was resorted to. This was no doubt due to psychic conditions. The patient was a young man, seventeen years of age, on whom a previous unsuccessful attempt at suspension had been made by another laryngologist.

In some of our earlier cases we had great difficulty in suspending patients on the first attempt, due to gagging and irritability. We explained that it was necessary to get the patient trained to this procedure, but now this seldom occurs. This is probably due to the fact that in all our earlier cases we used twenty per cent cocain for anesthesia, but recently we are using the pure flakes. The cotton-wound applicators are moistened by dipping them into the adrenalin and then into the pure flakes, then the application is made to the mucous membrane of the back of the tongue, pharynx and larynx, thereby obtaining a perfect anesthesia and muscular relaxation. We have no hesitancy in employing sufficient cocain to obtain the desired results; in some cases as much as four grains are used, and up to the present time we have never seen the slightest toxic results.

The apparatus which we employ is very simple and the instruments few. It is surprising how easily it can be accomplished with a simple outfit. An ordinary surgical table of any description that can be raised and lowered is suitable for attachment of the crane. Of course, the new table of Lynch's is preferable. The crane which was first devised by Killian is used, and we feel it answers the purpose satisfactorily. In the beginning, while using either the original Killian handle and spatulas or the modification of Albrecht's, we experienced great difficulty in keeping the tongue from slipping to one side, or the epiglottis slipping away from the spatula and thus obstructing the view of the larynx. Since using Lynch's modification these difficulties have all been practically overcome.

There still remained one great difficulty in this work, and that is where we desire to reach the vallecular region or to do actual work on the epiglottis. As all the spatulas with which I am familiar cover this area, I have suggested one which I believe will do away with this difficulty. It is a modification of a spatula, in which the tip is cut out in a crescent shape and the points reach well towards the pyriform fossæ, thus allowing the center portion to be free and permitting direct approach to the epiglottis and center of base of tongue.

A Cordes punch biting forceps, with its several sized tips, a Killian alligator bronchoscopic forceps, a long handled curette, and a few applicators for wiping way the little oozing, completes our list of instruments.

Probably the most frequent use of the instruments is for examination and removal of microscopic specimens in suspected malignant disease. Under suspension one can see the entire larynx exposed, and can study the limitation of the growth and very easily remove a portion for microscopic examination.

Owing to the fact that carcinoma cells are sometimes implanted in neighboring healthy tissues by cutting or snaring away some of the tumor, Bloodgood has recently called attention to the method of using a cautery needle by which he surrounds the specimen to be removed, thus believing he prevents an implantation in surrounding healthy tissue. As this needle is impossible to use in the larynx, I have suggested a specially long handled cautery knife, by which I cauterize an area completely around the specimen to be removed and thus obviate this danger of implantation, and the specimen can then be removed with impunity. So far as the interior of the larynx is concerned, the danger of implantation is not so great, owing to the fact that it is within a cartilage box.

Next in importance is the removal of the nonmalignant tumors of the larynx, especially the cords—viz., papilloma, fibroma, angioma, and polypi. It is a very easy matter to remove the tumor which presents itself in prominent view with a Cordes biting forceps. If the growth is situated at the extreme anterior commissure, it is sometimes necessary to use counter pressure externally, and this can either be done by an assistant or one can employ the left hand and thus easily bring the growth into view. In tubercular ulcerative laryngitis where actual cautery is indicated, or an application of trichloracetic acid is necessary, this can be done easily and far more accurately than when using the indirect method.

In fact, all of the conditions enumerated before can be done so much more satisfactorily than by the indirect method.

CASE REPORTS.

Case 1.—Master W., aged seventeen years. Patient has been complaining of hoarseness for the past two years, condition gradually becoming more pronounced, until now he can scarcely speak above a whisper. Has also had slight difficulty in breathing during the past two or three months. Family history negative as to lues and tuberculosis.

This is a case that I mentioned in which, although perfect cocainization was done, it was impossible to suspend the patient without a general anesthetic. He was referred by a laryngologist who stated that it was impossible by the indirect method to obtain the slightest view of the interior of the larynx. An attempt was made to suspend patient under local anesthesia by the same laryngologist, but it was unsuccessful. The young man was extremely nervous and greatly feared another attempt of suspension. Notwithstanding his fears, we attempted to suspend him, but it was impossible on account of the incessant gagging and struggling. Immediately he was placed under ether anesthesia, and there was not the slightest difficulty in suspending him satisfactorily.

Examination showed a large papilloma situated at the anterior commissure. This was easily removed with the Cordes punch. As soon as the patient recovered from the anesthesia the voice was almost normal and gradually improved until at the end of the fifth day phonation as well as breathing was perfect. I then attempted to obtain a view by indirect examination, but this was impossible. Wishing to see the larynx again, I made another attempt to suspend under local anesthesia, but found it absolutely impossible for the reasons described above. Microscopic examination showed the growth to be a true papilloma.

Case 2.—Mr. H. M. K., aged forty-four years, referred to us by a laryngologist from Fort Wayne, Indiana, with the following history:

Patient an inveterate cigarette smoker, but having had no illness of any consequence since childhood. He denies a luetic history. About one year ago the throat began to bother him; first noticed hoarseness, which increased, and was finally accompanied by slight pain and discomfort. Described the throat as feeling swollen. Was seen by a laryngologist in November, 1916. At that time he presented a congested condition of all the mucous membrane of the pharynx and larynx, and there was an active ulceration of the epiglottis and the left side of the larynx. The cord on the left side presents marked thickening and roughness along the free border. He gave a positive Wassermann, and the family physician was advised to give salvarsan intravenously and follow up with mercurial inunctions, and potassium iodid in doses of seventy-five to one hundred grains, three times per day. This was not done, as near as the laryngologist could ascertain; in fact, he believed the patient did not receive the salvarsan injection. Finally, the laryngologist gave him mercurials and iodids with a marked improvement in the general condition of the throat. Quite recently, however, the throat took on the appearance that it had when referred to us. A piece of tissue was removed for microscopic examination, but report was not obtained at the time he was sent to us. It was the belief that the original lesion was specific, as it had all the earmarks of the same.

Under local anesthesia the patient was suspended, and from the left arytenoid to the right, toward the epiglottis, there was a large mass causing a stiffening of the epiglottis. The entire left side was fixed. Three large pieces of the growth were removed for microscopic examination, and the same proved it to be a rapidly growing carcinoma. As there was no metastasis in the cervical glands, a laryngectomy was advised. The patient returned home and has not been seen nor heard from since.

Case 3.—Miss Hln., aged twenty-nine years. Referred by a laryngologist from Indianapolis. Has complained of hoarseness for several months. A diagnosis of a tumor mass on right cord was made by this laryngologist, and three unsuccessful attempts were made by the indirect method.

Upon examination I found a small apparently firm mass on the right cord near the anterior commissure. The patient was very nervous and feared the ordeal of a suspension, but after thorough cocainization she was suspended without any difficulty, and the entire growth was removed very easily. The patient did not have the slightest ill effect whatsoever. Her voice cleared up, and I have heard since that she is in very good voice.

Case 4.—Master P., aged five years. Referred by a laryngologist on account of hoarseness and difficulty in breathing. Wearing a tracheotomy tube. When patient was one year old, the parents noticed that his voice was quite husky; this condition gradually grew worse until he was three years of age, when his voice was completely gone. Consulted a physician, who did a laryngotomy and removed a growth from the larynx. Voice did not clear up and the difficulty in breathing continued. Two weeks later he was returned to the hospital and again operated, removing some more tissue. This time patient had so much difficulty in breathing that a tracheotomy was performed and tube inserted. One year later consulted another physician,.a laryngologist, who, after several attempts to examine the larynx, was unable to do so, and referred the patient to us. Examination shows papilloma extending completely around tracheal opening. The tracheotomy tube which he is now wearing has not been removed from trachea for one year.

On April 6th, under general anesthesia, patient was suspended and a large papillomatous growth was removed; this was situated just below the epiglottis. One-half of the external papillomatous growth around the tube was also removed. Child improved some, but on April 19, 1917, under general anesthesia, he was again suspended and the remaining half of the papilloma was removed and the interior of the larynx curetted. Small rubber catheter passed through laryngeal opening and out the mouth. This was left in situ to act as a guide in introducing the intubation tube on the following day, and then removed. Voice gradually improved, and the opening in the neck is gradually closing. The tube has now been left out for a whole week, and breathing is very good. We are gradually training him to use his voice.

Case 6.—Baby Smith. First seen May 20, 1915, aged three years. Complains of hoarseness and difficulty in breathing since she was five months old. Condition came on after a severe cold. Began to gradually lose her voice. This condition of her throat also interferes with eating. Healthy child otherwise. About one year ago was threatened with pneumonia, at which time had very labored breathing.

Examination showed a smooth neoplasm in the posterior commissure, anterior half of cords plainly seen, though quite red. Digital examination showed smooth round growth in posterior right laryngeal region.

May 24, 1915, under ether anesthesia, open drop method, a tracheotomy was performed. Trachea found to be small and easily collapsible. Suspension was applied, and from the posterior portion of the larynx a papillomatous growth was removed. Condition cleared up and patient returned home.

May 1, 1916, returned on account of recurrence of husky voice and difficulty in breathing. Under general anesthesia under suspension a small piece of papillomatous tissue was removed. The growth seemed very thin.

May 18th, under general anesthesia, an intubation tube was passed into the larynx of the patient, after the method of Iglauer, with a great deal of difficulty and serious symptoms of collapse upon the part of the patient, which required an emergency enlargement of tracheotomy wound and insertion of tube. The child was so bad—that is, breathing so labored—that the attention of a physician was required, and she was thought to have bronchopneumonia and was unable to take any liquid by mouth. Intubation tube was removed and tracheotomy tube reinserted, and patient had relief.

May 25th, patient well, pneumonia cleared up, and upon examination found that when the tracheotomy tube is closed up patient is able to enunciate a number of spoken words.

June 7th, patient was again placed under general anesthesia and both tonsils were removed, followed by an uneventful recovery.

June 10th, reentered hospital to have tracheotomy tube removed under observation. The fright was so great on the part of the patient that the attempt was given up.

June 22nd, tube removed and watched all day; nothing happened, and she got along very well. Tracheal opening healed over very nicely.

June 29th, patient left for home. Opening entirely healed.

On March 17, 1917, we received a letter from her father, stating that her voice is just as strong as any other child's, and she is talking all the time.

Case 7.—Mr. Msh., forty-six years old. Referred by laryngologist with the following history: One year ago, while in Mexico City, during the revolution, was frightened by an attack on his business, and in a fit of despondency he attempted to commit suicide by cutting his throat with a razor. Shortly after that was taken care of and the wound healed. He left Mexico and went to California, and while there noticed that his voice was gradually getting hoarse, and had some difficulty in breathing. A growth was removed from the larynx at that time by the indirect method. Some time later, however, this condition recurred, and this time he was suspended and another growth removed, which he informed us had been microscopically examined, and was granuloma.

September 13, 1916, patient first consulted us, and by indirect examination a smooth looking, subglottic, sesile growth was seen. Cords looked normal.

September 18th, under suspension, local anesthesia, this soft growth was removed with the Killian short alligator forceps. Subsequently curetted and cauterized the base with a twenty per cent solution of silver nitrate.

September 27th, throat looks good. Breathes well. Microscopic examination proved this growth to be nonmalignant.

October 11th, no recurrence; in good condition. Discharged.

Case 8.—Mr. Mstrs., fifty-four years of age. This last case that I desire to report is the one in which suspension was easily performed, but the growth could not be reached. Dr. Beck reported this case before as a case of adenocarcinoma situated on the left vocal cord, near the anterior commissure. Massive doses of X-ray treatment had been given, which seemed to cause it to disappear, as no sign except a roughness of the cord could be seen six months after the discontinuance of the X-ray.

He returned one year later, and what seemed to be granulation was removed at site of old growth. This was curetted away and the base touched with silver nitrate and patient placed under observation. Examination about four weeks later showed a growth on the laryngeal surface of the epiglottis. He was again suspended and an attempt was made to remove a portion of this growth, but owing to the pressure of the spatula against the under surface of the epiglottis, this was absolutely impossible. This was again attempted, but unsuccessfully, as the spatula prevented the under surface of the larynx from being brought into view.

Under local anesthesia an exploratory incision was made over the larynx. Beneath the cricothyroid membrane a neoplasm was observed which had grown outward through the larynx, but which seemed to be encapsulated. Knowing the nature of the original neoplasm, we decided that nothing short of a laryngectomy would suffice. Consequently, three days later, under local anesthesia, a laryngectomy was performed, and patient made an uneventful recovery, and thus far there is no recurrence, four months since operation.

LIII.

THE DIAGNOSIS OF CEREBELLAR DISEASE—REPORT OF TWO CASES.*

By Edward J. Bernstein, M. D.,

Detroit.

The diagnosis of cerebellar disease, more especially of cerebellar abscess, is of vital interest to otologists whose sphere of activity lies in such close proximity, as the etiology bears so close a relationship to ear suppurations.

The subject is not readily comprehended and is still a matter of much conjecture, but there is much opportunity for putting into practical use what information can be gained by study. Much of the difficulty in the past in considering this abstruse matter has, it seems to me, been cleared up by a very simple key which in nowise alters the data as given by the Vienna school. This key is from the pen of Dr. Isaac Jones of Philadelphia. Alfred Braun, in a recent article, spoke of some eighty-six cases which have been reported since 1897. I am certain, as I look over the histories of some of my mastoid cases, I have in the past seen at least five or six cerebellar abscesses which were unrecognized on account of the lack of the knowledge which we now possess.

Cerebellar abscess is almost invariably due to ear suppurations. It is not always single, but may be multiple, and complicated with subdural or temporosphenoidal abscess. It may come with apparently no visible lesion in the middle ear—such cases have been seen and reported. It may be chronic or acute, encapsulated or free. One of the unfortunate conditions in connection with cerebellar abscess is that it often runs its course with practically no clinical symptoms, and death may occur before any symptom of intracranial involvement can be detected. It is often obscured by other otitic or intracranial conditions. If encapsulated it may remain latent, quite unsuspected, for quite a long period; such a case is reported in Phillips' book.

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*Read before the Detroit Oto-Laryngological Society, January 16, 1917.

An abscess in the antral or tympanic tegmen may lead to temporosphenoidal abscess. In the posterior group of mastoid cells or in the labyrinth there is a tendency to cerebellar abscess. The clinical course generally shows itself in three stages. In the initial stage, which more often follows a chronic than an acute abscess of the middle ear, the symptoms are not sufficiently severe to arouse suspicion. There may be some rise of temperature, headache, more or less severe, and vomiting. These are, however, of short duration, and seldom arouse suspicion of the seriousness of the condition. The disease runs a rapid course. One should be on guard for cessation of otorrhea, which often means the onset of intracranial complications.

There may be a latent stage, in which a very considerable brain or cerebellar abscess may be present, and may cause no definite symptoms, except probably a complaint of moderate headache and intracranial pressure, especially on increased mental effort. As the abscess progresses headaches become more or less severe and more or less localized, but their localization may bear no relation to the site of the abscess. Indeed, the symptoms may indicate trouble on the opposite side. Neither does palpation or percussion give any definite clue. In cerebellar abscess, in the early stage, frontal as well as occipital headaches occur. In my last case, to be related hereafter, the patient complained only of intense pain in the nape of the neck. Loss of appetite and loss of weight possibly, in the beginning, acceleration of the pulse rate, may be present, and slight rise in temperature marks its onset. Finally there come distinct symptoms referable to the brain, ushering in the manifest stage. Symptoms of septic absorption are now apparent. Greatly increased leucocytosis, increased headaches, general or local, and more or less expulsive vomiting point to the trouble. Optic papillitis may occur. Its absence does not influence diagnosis; indeed, it is more often absent than present. As intracranial pressure increases the temperature becomes subnormal and rarely rises, the pulse slows down to fifty or sixty, there is impairment of mentality, various forms of aphasia supervene, later somnolence and apathy. alternating with excitement, follow.

In cerebellar abscess and new growths, patients who are able to walk show a characteristic gait (cerebellar ataxia). There are oculomotor paralysis, mydriasis, ptosis, facial palsy and abducens paralysis. Rigidity of the neck is one of the late symptoms of cerebellar abscess. If not relieved death ensues from increased pressure on the respiratory center. The respiration is slow, irregular, often of the Cheyne-Stokes variety, or the patient dies rapidly by sudden cessation of respiration, by rupture into a neighboring ventricle, or a meningeal involvement, with high temperature, rapid pulse, vomiting, convulsions and death.

Spontaneous recovery may occur by encapsulation of the abscess. Cerebellar abscess is oftentimes associated with sigmoid sinus thrombosis. The disease usually lasts from one to three or four months. In the latent stage, when the abscess involves motor centers in the cerebellum which are known to enervate definite movement, it had seemed possible that we might say, after full examination of the various signs, that the abscess or growth, as the case may be, occupies a certain definite portion of the cerebellum. A late analysis of some thirty-one cases of cerebellar tumor and abscess, by Dr. Ernst C. Gray of Boston, in the service of Dr. Cushing, shows that much which we had previously supposed would lead us to definite localization is not nearly so securely based, and without being able to exactly localize, all we can now tell is whether it lies anteriorly or posteriorly in the cerebellum, or probably whether left or right.

In considering localization of cerebellar growth or abscess we must recall the fact that the cerebellum records sensory impulses and sends out motor impulses, and the motor nuclei in the cerebellum are closely connected with the vestibular nuclei. Motor impulses reach the cerebral cortex from the cerebellum through the superior peduncle. Motor impulses from the arms, limbs, and other muscles reach the cerebellum through the inferior and middle peduncles. These motor impulses are met by sensory impulses from the muscles and joints, and by impulses from the vestibular apparatus, and are here acted upon in such a way by the centripetal impulses as to make them effective in maintaining the equilibrium of the body. Any interference, either by abscess or by growths in these tracts, or in the nuclei or in these centers which have been found to enervate the given motions in the various joints, must necessarily disturb this equilibration. By dint of long continued research it has been ascertained that behind the internal auditory meatus, in the lobus biventer, there are distinct centers for the shoulder and wrist. Certain other centers for the motions of the shoulder have been found in the posterior inferior lobe. Certain other centers have been found for the motions of the trunk and hip muscles. It seems logical then that when a disturbance in the equilibrium of these joints occurs, not due to evident external causes, that we could say the disturbance lay centrally in the given centers thus ascertained.

Ordinarily a normal person with his eyes open, having pointed at a given object, can again touch the same object with his eyes closed. A patient with a disturbance in the cerebellum involving those centers enervating the joints thus used will not be able to do this with his eyes closed, and in attempting to point will deviate away from the object on the same side as that in which a cerebellar lesion exists. When a normal individual is rapidly turned in a smoothly turning chair ten times, and then suddenly stopped, he can no longer point true, and he imitates the condition of an individual with a cerebellar lesion. If a patient, on being turned, instead of deviating, points true, it indicates a cerebellar lesion.

Nystagmus is another one of the phenomena of cerebellar lesions. We must remember this fact, however, that nystagmus always means one of three conditions: either it is ocular, in which case it is always regular, rhythmic, and not influenced greatly by direction of gaze, and is usually due to a lesion in the eye capable of demonstration, except in such forms of nystagmus from congenital troubles which have been overcome, or in occupational disturbances such as miner's nystagmus. There is also a perfectly physiologic nystagmus in some individuals on directing the gaze to the extreme right or left. Nystagmus of cerebellar or labyrinthine origin differs from this in that it is of an entirely different character, being composed of a quick (cerebellar) movement and a slow (vestibular) movement, so that in the presence of these latter phenomena, especially when it exists spontaneously, we must conclude that we are dealing either with a labyrinthine or cerebellar condition. This form of nystagmus can be evoked by rapidly turning the patient in a chair; by the caloric, galvanic or fistula tests, in which instance it has distinct characteristics both for the normal and for the pathologic conditions.

The difficulty of remembering these various reactions as given out by Bárány has deterred many from studying these phenomena. It seems to me that the key given by Dr. Jones, of which I spoke, while not altering the basic principles of Bárány and the Vienna school, do make it easier to comprehend them and to put it into practice. In brief it is as follows: A normal person when turned in a chair to the right will develop this nystagmic movement of the eye. Instead of considering it in terms of a quick and slow component, it is best to note the slow movement, which is a vestibular one and always in the direction in which a normal patient is turned. Also this same normal individual will now deviate in pointing to the same side to which he is turned. Applying cold water (temperature of 60° F.) to the normal ear will cause the eye to deviate to that same side, and the patient will again deviate in pointing away to that same side. In other words, turning a normal person to the right, a slow movement of the eye to the right is observed and a pointing deviation to the right. A cold caloric to the right ear will cause a slow deviation of the eye to the right and a pointing deviation to the right. Any changes in these phenomena indicate a lesion either in the labyrinth or the cerebellum or one of its tracts.

Labyrinthine nystagmus of diffuse labyrinthitis cannot be distinguished from cerebellar nystagmus in the first examination. Subsequent examination will show that the labyrinthine nystagmus tends to disappear by compensation. On the other hand, if it is due to cerebellar lesion it remains constant or becomes worse. When in such a case we can show that the labyrinthine function is destroyed, when the eyes deviate, either spontaneously or under stimulus, away from such an ear, it is obvious that the trouble is retrolabyrinthine—i. e., cerebellar. In a patient with nystagmus the direction of which changes suddenly, one may look for cerebellar involvement.

Hypermetria, unmeasured or immoderate movement, may occur spontaneously, but more likely in commanded movements done quickly.

Another symptom which may be present is cerebellar asynergy—inability to make the trunk follow foot movements, resulting in arrested progression. With arms folded the patient cannot rise from bed while lying on the back.

Adiadokokinesis is a loss of ability to rapidly execute successive movements. Muscular force is intact, but combined successive pronation and supination is more slowly executed than in the normal.

Tremor. On pointing to an object, orientation is correct, but movement is not uniform; there are oscillations in various directions.

Disturbances in writing. The patient being requested to make a dot on paper with a pencil, either goes through the paper, breaks the point, stops before reaching the paper, or makes a line instead of a dot. In attempting to make the round letters, like C and O, he gives them polygonal shape instead.

Disturbances of speech. These may be scanning, jerky, a little explosive, sometimes draggy, similar to disseminate sclerosis.

Atony or hypotony may be unilateral or bilateral. When the former, it is upon the same side as the lesion. This symptom is brought out by having the patient flex the forearm forcibly, the examiner opposing it by traction. When suddenly let go, in normal persons a pronounced flexion occurs, followed by violent extension.

Pointing reaction. We have come to a definite conclusion that all pointing reactions are primarily cerebral and not cerebellar, as hitherto stated.

Dr. Jones rightly maintains some inconsistency of findings, even in the same case, marks the vital characteristics of our subject. We are dealing with advancing processes, not fixed morbid lesions.

In reference to the apparent overthrow of our accumulated evidence regarding cerebellar localization. in a personal letter

Dr. Jones has this to say: "The main value of the ear tests seems to me to be, first of all, the rounding out in diagnosis of ear conditions, and then the definite value in finding out the cause of vertigo in any given case. They usually clear up well under these tests, but from the standpoint of the neurologist and the surgeon, in locating intracranial lesions to be sure there is a great deal that is encouraging from the development of ear tests. As for cerebellar localization, as a rule the cases (autopsies and operations) have shown rather accurate results of ear tests—not only whether the cerebellum is involved or not, but also which hemisphere, whether posterior or anterior, is involved. They have been particularly helpful in determining whether the lesion is operable or not."

Case 1.—On June 28th, this past year, I was asked by Dr. V. of Kalamazoo, to see a young man of twenty-nine years, who had been suffering for many years with running ears on both sides. The right ear discharged almost continuously, and the left more or less constantly. For the past year he had considerable pain in the mastoid region of the right ear. A few weeks before I was called he saw a specialist, who correctly insisted upon a mastoid operation, but this was refused, and he consulted his family doctor, who removed a polyp from the external auditory canal. The specialist who saw him told me that the man was suffering great pain and could hardly maintain his equilibrium and was very dizzy, and he made the tentative diagnosis of labyrinthine involvement, which was doubtless correct as far as it went. At any rate, when I saw him he was suffering excruciating pain in the nape of the neck and nausea, with a pulse rate of sixty and temperature of 99.2°. There was no tenderness over the mastoid, but a foul bloody purulent discharge from the right ear. It was impossible to get him to raise the head on account of the nausea and dizziness. Hearing in the ear was gone.

I was unable to make the proper tests for differentiation of labyrinthine and cerebellar abscess, but the clinical signs were so unmistakably of brain abscess and case so urgent, that he consented to immediate operation and was taken to the hospital and operated on at 9 p. m. A radical was done, and not a single mastoid cell was found in the ivory hard bone till we reached the antrum, when a large cholesteatomatous and fungoid mass was removed; the tegmen antrum was carious, and further chiseling opened a large subdural abscess. I felt then that there was most likely a cerebellar or cerebral abscess, but hoped that this would drain enough. At any rate, I dared not do more, as his condition was rather critical, although I was not more than forty-five minutes doing the operation, despite the ivory bone. The patient was dressed and returned to bed, the family being informed that though I had found pus in the mastoid and some in the cranium, there was great likelihood of more being found deeper, and that another operation might be called for in a few days.

The patient passed a comfortable night, the first in weeks. His temperature next morning was 98.2°, and pulse eighty. He continued comfortable all that day till about 9 p. m., when he again complained of the pain in the nape of the neck, and his pulse rate was again sixty. I tried some of the more readily applied tests for cerebellar trouble, and felt sure we were dealing with a cerebellar abscess and so informed his physician, who opposed further operative procedures. During the following day the symptoms of cerebellar abscess were more pronounced, and, in spite of my best endeavor, I could not make the family doctor see otherwise but that the man was better. It was only after I threatened to withdraw from the case and explain my position to the family that I obtained the tentative promise to let me operate next day, if he thought the man was not better. It was a fatal mistake to even concede that much, as subsequent events proved. I tried to get consent to operate next morning, but was again blocked and promised a trial at noon, the doctor still insisting that the patient was better, but at noon I found him almost in extremis, under which circumstances the attending physician finally consented to operation. The patient died on the table while I was about to open the original wound. He died of respiratory failure—typical of untreated cerebellar abscess. Fortunately, I obtained a postmortem examination, and it showed general leptomeningitis and a large abscess in the anterior portion of the lobus biventer of the right side. There was also a small abscess under the anterior side of the left side. There had been no optic papillitis on either side.

Case 2.—On August 16th I was asked to see Wm. M., aged twenty-four years, on account of loss of sight. He had absolutely no light perception in either eye, with very marked choked discs on both sides. The nerve was about three diopters high on left side, and about two on the right, where there was beginning optic atrophy. He had a slight facial paresis on the right side, exophthalmos on each side, no sign of enlarged thyroid, but he had a very high pulse rate which was easily increased by slight effort. He had a fine vibratory tremor on extending the hands, as in Graves' disease. There was a slight Romberg but no Graefe symptom. As he had no light perception, an Argyl-Robertson was out of the question. He had marked rotary nystagmus (spontaneous) on direct gaze, which was increased when he looked to the left, and not markedly increased when he looked to the right. He also had a slight vertical nystagmus when he looked up, but not on downward direction of the eyes. His trouble began two years before, with spells of falling backward. These have continued with more or less persistency ever since.

Spontaneous pointing tests, from the shoulder, wrist and elbow always deviated to the side of the arm tested—that is, to the right when the right arm was tested, and to the left when the left arm was tested. Heart was normal in size, but as well as I could make out there was a peculiar reduplication of the first sound.

Turning him to the right in the chair produced nystagmus to the left (Bárány nomenclature), but as I think we now will call it deviation to the right, and during this he pointed correctly.

Turning to the left produced deviation to the left and pointing to the left. There was presumably no labyrinthine trouble present. My diagnosis was a probable cerebellar growth. When observed again, about a week later, he had no rotary nystagmus, but a slight horizontal spontaneous nystagmus to the right was seen when he looked straight ahead; on looking to the right, slight increase of right nystagmus. On looking to the left he had a marked increase of the nystagmus to the left.

Pointing test. On turning him to the right he deviated to the right; on turning to the left he pointed true. Spontaneous pointing, shoulder test down, was true on the left side but deviated to the right in upward movement of the left arm. On the right side spontaneous shoulder reaction from below up, deviated to the right; from above it deviated to left. Cold caloric to the right ear gave a horizontal deviation, slow vestibular movement, to the right; cold caloric left ear gave deviation to the left. Hot caloric in left ear accentuated the deviation to the left; on the right side it was negative. Pupils of both eyes were uniformly dilated to five millimeters and did not react to convergence—and of course not to light, as he had no light perception. The nerve was markedly choked in the right eye, with very tortuous veins, and arteries hardly perceptible. Hypometria right normal; only fairly executed left; no cerebellar asynergia; slight adiodokokinesis on the left side; tremor bilateral; no disturbance of speech apparent; some hypotony. In extending the arms out straight, he holds the left one lower than the right.

It was thought from this symptom complex that we had to deal with a cerebellar new growth—syphilis being excluded by a negative Wasserman. It was thought that we would find a growth somewhere in the region of the cerebellopontine angle, but most probably in both sides of the lobus biventer, according to the comparison of these findings and Bárány's results. The work of Gray supports the more conservative opinion of Dr. Biefeld, who simply stated that it is somewhere in the cerebellum. This is still a great help in case of operative procedure.

Owing to the fact that there was now very little hope of restoring his sight, and the very great probability that the patient might die from the anesthetic alone, in view of his heart condition, I could only state the chances and my willingness to undertake the operation, provided the family were willing to stake all or lose all, and they wisely decided not to take the chance.

SOCIETY PROCEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY,

Meeting of April 13, 1917.

Exploratory Tympanotomy for Relief of Deafness.—Presentation of Two Operated Cases.

DR. HAROLD HAYS said that, like many others, he has been studying the question of adhesive processes in the middle ear, and whether we would be able to get at these adhesive processes by operative procedure. We meet with a number of cases with fairly wide open eustachian tubes and retracted drums, cases in which we can do practically nothing so far as hearing is concerned. Many of these cases occur in young persons between twenty and thirty years of age. In about thirty per cent of these cases we have prolonged bone conduction, indicating some interference with the transmission of sound waves. This is probably due to adhesions between the ossicles or between the drum and the internal wall of the middle ear. Dr. Hays said, further, that having had this in mind for some time, he was finally able to secure some fresh autopsy material, with which one is naturally able to do work that is impossible with the ordinary cadaveric material, owing to the shrinkage and destruction of tissues. With this material he had been able to devise a practical operative procedure which is simple, yet apparently effective.

The patients presented (two) were operated upon only a week ago, and it was not yet possible to state whether or not the hearing would be permanently improved, though there was strong hope that it would be. Neither one had had any reaction whatever—no fever, nausea, nor vomiting—within the first few days. On the fifth day, however, the young man had become nauseated, but that cleared up under calomel. Both patients' wounds healed by primary union, and there was nothing to be feared from an active labyrinthitis or secondary infection, though both still have some slight discharge.

Of course, it was impossible to make any hearing tests at the present time, but neither of the patients could hear a watch or the whispered voice before operation, and both claim that they can now hear much better with the operated ear than they could before.

Dr. Hays said that he offered this procedure merely to show that it is practicable from an operative point of view, and that he hoped to employ it in a great many more cases. It was desirable that the test for hearing should be made by someone else before the operation, who would repeat it some time after the ear becomes dry, and note the result. If the procedure accomplishes what is hoped, the hearing will be improved in an appreciable degree.

Someone had asked how it was expected to prevent the formation of other adhesions. To that one might reply as the abdominal surgeon does. He does not claim that other adhesions will not form, but he endeavors to prevent them from interfering with function. Everyone has seen cases where the middle ear has numerous adhesions and yet the hearing is good.

DISCUSSION.

Dr. Seymour Oppenheimer said he hoped that the work which Dr. Hays had been doing would not prove to be love's labor lost; but the same work had been attempted by himself according to a different technic, and it had been tried by others, dating back long before his time or Dr. Hays' time. All are familiar with the very delicate instruments which were designed and used by Buck, Roosa, and others for the severing of intratympanic adhesions; yet these have long since been forgotten and fallen into disuse. One point Dr. Hays has lost sight of, and in all probability all other workers as well, is that the ossicular chain is not necessarily the only portion of the auditory apparatus that is concerned in the question of hearing, and it is not alone adhesions that form about this portion of the ossicular chain, but adhesive fibrous tissue forming over the promontory and about the oval window, binding down the stapes, which interferes with the sound wave and is the most responsible for deafness. The question of whether or not an individual has a drum does not always influence the hearing. Patients are often seen with almost no drum remaining as the result of a suppurative process, yet with almost normal hearing.

Some fifteen years ago Dr. Bates, an ophthalmologist of this city, advocated cutting away the adhesions around the oval and round windows on the promontory. After his preliminary operation he found that the hearing of the patients was very decidedly improved; but he was compelled subsequently to reoperate repeatedly in order to maintain the hearing. These operations were done under local anesthesia. In one instance he operated no less than seventy-odd times, and each time that he cut away the fibrous connective tissue there was a definite improvement in the hearing, which, however, always reformed and the hearing receded to where it had been before.

Stimulated by Dr. Bates' suggestion in this line of work, Dr. Oppenheimer took up the proposition, some twelve years ago, and attempted, after having performed the Stacke operation, to cut a trephine opening in the promontory, for which purpose he devised some very delicate instruments, and then attempted to transplant over the opening which he had made something which would act and allow the transmission of the sound wave as the drum membrane would, employing cargile membrane, then areas of epidermis, the vitellum of the egg, etc. He succeeded with the epidermal graft in a number of instances, and while in each of these cases there was a very definite improvement of the hearing for the briefest time, yet it was not maintained.

While Dr. Hays' work is very admirable, it was to be feared that his final results would be disappointing.

Dr. Hugh Blackwell said that while he hesitated to speak in a derogatory manner of work that was still in its incipiency, anyone who attempts this work should bear in mind the ever present danger that in these cases the patient may develop a mastoiditis. Of course, both these patients have now what is practically an acute inflammation of the middle ear. A number of years ago Dr. Heath treated the drum with cantharides and produced a very severe reaction. In one instance the patient developed an otitis media purulenta acuta and had a series of complications. Dr. Blackwell said he had also heard of another case which ran through numerous complications and developed an acute otitis, and finally died of miliary abscess.

Dr. Haskin said that Dr. Hays was to be congratulated upon attempting to do almost impossible things, but that it was nothing new to break up adhesions. Work of this kind was described in Politzer's old book. It is simply a question of getting at the adhesions, wherever they may be. As he understood, Dr. Hays goes behind and separates the membranous canal from the bony, thus reaching the middle ear. As Dr. Oppenheimer had said, one must feel some pessimism as to the final results. Most otologists have tried procedures of this kind, and all have failed. Dr. Yankauer had experimented, making incisions in the drum membrane, and had failed. Dr. Haskin said that he himself had tried it in a series of cases, and had failed. It has been impossible for him to keep these adhesions from reforming. He hoped that Dr. Hays would succeed, but feared he would not. Dr. Sexton had tried to do the same thing, and for a time there was great enthusiasm all over the country about his work, but finally he had to disappear on accountof damage suits resulting from the effects produced. Apparently, the hearing is improved for a while, and then it grows worse.

Dr. Hays (closing the discussion) said he thought everyone present would appreciate the fact that he was not claiming to be attempting to do anything new so far as the middle ear is concerned; he was merely trying to find a proper approach to the middle ear, so as to get a certain exactness which cannot be attained by the ordinary method. All must admit that work in the middle ear through the canal is a very inaccurate procedure; moreover, the attempt to work in the middle ear through any such opening as given by the Stacke operation is unsatisfactory also. By this new operation one gets a very fair view of the middle ear. The illustrations shown were made by an artist who had never before seen the middle ear.

He had thoroughly reviewed the literature on the subject, including Politzer, etc., and had noted, moreover, what Politzer states, that if an opening is made in the ear drum, as long as it remains open, the hearing will be better. But we have never been able to keep a perforation of that kind open permanently. Again, Politzer, like others, tried to relieve adhesions, and cut the muscle leading to the malleus, and the stapedius muscle, etc., but not with any particular exactitude. Dr. Hays said that so far he has only adapted eye instruments for this work.

As for the work done by Dr. Bates and others, Dr. Hays said he believes that if one attempts to remove any definite adhesions around the oval window he would fail—one does it in a way that he is bound to have adhesions; but there are any number of these cases where the adhesions in the round and oval window can be modified. The procedure was presented merely as an operation which might prove successful in a number of cases.

Replying to Dr. Blackwell's remarks, Dr. Hays said that any patient upon whom this operation is performed should have so little hearing that he would be willing to take any chance for improvement; patients like those presented, who cannot hear at all, and cannot work because of deafness, will be willing to take great chances. The fear of a possible labyrinthitis in these cases had worried him a good deal for a few days. Another point which had been brought up was the question of the Heath treatment for dry relaxed ear drums, which was a very different thing from adhesions in the middle ear. Dr. Hays said he had written a paper on this subject some time ago. Dr. Hays said he had never had any trouble in that operation. He had not had very many cases; but in the few cases where he had gotten results, they had been moderately good, and had been attended with no danger whatever.

A Case of Mastoiditis in a Diabetic. Extradural Abscess, Perisinus Abscess. Recovery.

Dr. Otto Glogau: Dr. R. H. Urquhardt (*Medical News*, March 21, 1896), in discussing his two cases of mastoiditis in diabetic people, said: "That there does seem a special tendency to inflammatory changes in the mastoid region in the dyscrasia under discussion, there is unmistakable evidence, but as to the existing relationships we are as much in the dark as we are ignorant of the true nature of the diabetic diathesis."

J. E. Sheppard (*Medical News*, May 2, 1896) does not believe diabetes to be an etiologic factor in mastoiditis.

A. H. Buck (Transact. Am. Otol. Soc., 1895, quotes the entire literature on the subject then existing, and is very much in favor of an early operation. The prognosis of mastoid operation on the diabetic is a good one in his opinion.

J. D. Richards (*Archives of Otology*, 1907) establishes even a pathognomonic symptomatology for diabetic mastoiditis. The slow march of the mastoid symptoms and the absence of marked inflammatory phenomena (until late) argues, according to him, not so much a virulence of infection in certain cases with extensive destruction, as a low vitality of the tissue. The postoperative condition of the wound also points to this. A successful issue depends largely upon the rapidity of operation and the short duration of the anesthesia. The bone should be removed well beyond the apparent limit of involvement and a very thorough operation done, as the tissues are suffering from a dyscrasia and are more extensively involved than is indicated to the eye. Diabetes seems to be accompanied by vascular changes. In diabetic sinus thrombosis the vessels should be opened but the clot not disturbed, except removing the main portion of the thrombus.

The literature on the subject became quite large of late. The question as to the etiologic importance of diabetes has not yet been settled. We know, however, on the other hand, that in many cases with normal metabolism, glycosuria was found after mastoid operation.

The writer operated four cases of mastoiditis in diabetic persons, including the one represented tonight. He comes to the following conclusions: Diabetes as such is no etiologic factor for mastoid involvement. The exciting cause is always a predecessing cold or other infection. In an ear thus weakened, diabetic mastoiditis develops. It is similar to the luetic changes within the ear and nasopharynx, which, according to Gerber and Gradenigo, also occur almost exclusively in previously affected organs. The pathologic process is very destructive and involves with predilection the sinus. The symptoms do not differ from other middle ear and mastoid suppurations. The operation should be performed very early. The narcosis should be short and consist of chloroform. The operative procedure should be as quick as possible. The diseased tissue should be removed further into the healthy portion than apparently necessary. The wound should not be closed. The percentage of sugar is of no importance as far as the prognosis is concerned, which in most instances, when the operation is performed early, rapidly and thoroughly, is a good one.

Following is the history of the case presented:

Mrs. C. R., fifty-four years old, took sick on April 7, 1917. She felt a slight pain in her left ear. A few days previously she had taken cold. Upon application of hot water bags the drum opened by itself. She was treated for an entire month by a doctor. Her condition grew worse. She developed severe pains over the mastoid and could not sleep any more.

June 3, 1916, I was called in consultation by Dr. Maguire, whom the family had asked to see the case. The patient was in a very much weakened general condition. There was a large swelling behind the right ear, showing redness and fluctuation. The entire mastoid region was extremely painful. The drum could not be inspected, as the canal wall was sagging to such an extent as to fill out the lumen entirely. There was slight discharge from the canal. The patient was suffering from diabetes for several months, and the sugar amounted to six per cent of late. Immediate operation was advised. The patient was removed to the Philanthropine Hospital. After the usual semilunar incision pus was seen in large quantity, and a fistulous opening was detected in the cortex. The mastoid process, including the tip, proved to be totally destroyed and replaced by necrotic granulations and pus. The necrotic process had reached the dura and formed an extradural abscess, which was opened and the dura exposed about one and one-half centimeters. There was an exposure of the sinus three-fourths of an inch in length, the destructive process having destroyed the bony wall of the sinus to this extent. The sinus wall seemed discolored, but not thickened. Under usual precautions the sinus was incised and a small thrombus was washed out with the current of blood. The bony tissue was removed away into the healthy portions. The wound was left open and drained. The narcosis, chloroform, lasted twenty minutes. The healing process was rather sluggish. The granulations were stimulated by scarlet red applications and dietetic measures. The external wound closed six weeks after the operation. The patient has now about two per cent of sugar.

Perisinus Abscess With Apparently Normal Sinus, Followed by Extensive Thrombosis With Multiple Metastatic Abscess. Removal of Clot From Sinus and Bulbus. Jugulectomy. Recovery.*

By Otto Glogau, M. D.

DISCUSSION.

Dr. Alfred Braun was inclined to question one statement made by Dr. Glogau—i. e., that in diabetic cases the operation should be as thorough as possible. The diabetic is a bad operative risk at best, and in the cases which he himself had operated on, the more thorough the operation had been the worse the result. The only cases which had recovered were those where as little as possible was done

Dr. Seymour Oppenheimer said that he had performed a number of operations on individuals with some glycosuria, but all the successful cases were operated upon under local anesthesia. Two cases that were operated upon under general anesthesia succumbed, though he did not know whether that was an important point or not. The general surgeon operates on many such patients who do not succumb. The question is not so much in regard to glycosuria as of the presence of diacetic acid or an acetonuria. Diabetics, of course, have very low reparative powers, and the destruction of tissue is very rapid. In one instance a distinct crepitus was felt on palpating the mastoid. Later it was found that by the pressure the cortex had been fractured.

The preoperative preparation of these patients is most important. Unless the case is showing some very violent symptoms, it is better to delay the operation for a number of days until the patient's condition has been improved by dieting. The administration of atropin will reduce the sugar very materially. The postoperative care of these cases is also very important—not so much the restriction of the diet, for that follows naturally after operation, but the administration of alkalies, both by mouth and per rectum, is of great value.

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*See page 788.

Dr. Oppenheimer said he agreed that the operation should be performed as rapidly as possible, and the operative work limited simply to the performance of proper drainage.

In the case of sinus thrombosis he had the privilege of seeing the patient in consultation with Dr. Glogau. She had evidently had an aseptic lower end of the thrombus, or the blood culture would have been positive. The question had arisen as to the advisability of primary jugular ligation. Dr. Oppenheimer said he objected to the primary ligation, as he does not consider it advisable before exposing the sinus. Then one can be guided by the condition as to whether or not jugular ligation should be performed. While it is true that probably ninety per cent of patients with a thrombus of the sigmoid, with a positive blood culture, do not require jugular ligation, yet some ten per cent at least can be handled without such a procedure, and he doubts the wisdom of beginning with a primary ligation. One thing that was very characteristic in this case was the absolute euphoria which this patient had; that is very characteristic of sinus thrombosis.

As to the question of the advisability of the surgical technic which Dr. Glogau employed: it is very doubtful whether the Gruenert operation is ever necessary. It may be necessary in this operation, by reason of certain anatomic conformities, to remove the condyloid process, but it is a question if the same result cannot be accomplished without resorting to that extremely difficult procedure. Having the torcular end of the sinus above compressed, and having the portion of the vein above the clavicle ligated, if it is not possible to establish free bleeding from the bulbar end of the sinus, one might content himself with introducing a drain into the bulbar end of the sinus and utilizing the upper end of the vein in the neck as it emerges from beneath the mastoid, as a drainage tube. He questions how much systemic absorption arises from the small collection of thrombus which is left in the bulbar end of the sinus, for such absorption can only be through the inferior petrosal sinus, which is of very small caliber. This thrombus disintegrates and is in the nature of a local abscess which drains into the neck wound. He believes this procedure simpler and just as efficacious as the difficult surgical proposition which Dr. Glogau employed.

Dr. Haskin said that he had had some experience in operating for mastoid conditions in diabetic patients, and has no hesitation in doing so. Dr. Oppenheimer had spoken of the importance of the postoperative care and alkalin treatment of these cases, but personally he had not found this necessary. In three of these cases he has given morphin and atropin before operating, and all recovered, apparently without any disturbance whatever; nor did any of the patients receive alkalin postoperative treatment, or any attention to the diet other than restriction.

He agreed with Dr. Oppenheimer in regard to the Gruenert operation, which is a beast of an operation. Anyone who will study dissected bones, as had been done at the last meeting of the section, will realize that in a majority of cases one can trace the lateral sinus and the sigmoid sinus through the mastoid and reach the bulb, for the external wall of the bulb lies directly in front of you, as it rises at the final turn of the sigmoid. In many instances it will extend under the middle ear, and you can get in by following the wall deep into the canal. You do not have to go through the deep soft tissues at all to get to the jugular bulb itself; the drainage from below, as described by Dr. Oppenheimer, is ample, and instead of using a drainage tube, chromicized catgut can be employed, as it is flexible and will be easier inserted into the vein.

Erysipelas as a Complication of Mastoid Operation.*

By Harold Hays, M. D.,

New York.

DISCUSSION.

Dr. Dwyer said he had not much to add, excepting to emphasize some of the points brought out by Dr. Hays. As far as the organisms are concerned, in all the cases that developed at the Manhattan Eye, Ear and Throat Hospital. the streptococcus hemolyticus had been found. They had never had erysipelas with mastoiditis that had any other organism.

As to the spread of the disease in front of the ear, that is probably because the lymphatics spread forward.

In making a differential diagnosis between sinus thrombosis, meningitis, and erysipelas, it is generally found that with erysipelas there is a low pulse, whereas in meningitis there is a high pulse—say a case with a temperature of 105°—within twenty-four hours after operation, and nothing else, but running a pulse of one hundred and twenty to one hundred and thirty. In such a case he had made a diagnosis of fulminating otitic meningitis rather than erysipelas, and the patient died later of meningitis. Dr. Dwyer said he had never seen a pulse in erysipelas go above one hundred. That is one good distinguishing point.

Regarding blood cultures, in uncomplicated erysipelas, the blood culture is negative. If the erysipelas lasts long enough, you can get a positive culture. In one case that he had seen, the patient had been sick for three or four weeks—the erysipelas would extend down to the feet and then clear up, and then break out again—and in this case a positive culture was obtained, and later death ensued. The longer the erysipelas lasts the worse it is, unless something like the leucocytic extract can be used.

Clinically, there are two distinct types of erysipelas. One occurring in strong adults, the sthenic type, where the temperature rises and continues high, like pneumonia; it continues for five to nine days, with a temperature of 105° to 108°, and then falls by slow crisis. The other type occurs in the asthenic individual, and it occurs and recurs in the same area from time to time, with a typical septic temperature of 100.5°—high up in the evening—totally different from the type that approaches pneumonia.

The blood count is useless, for it will not differentiate meningitis, pneumonia, or sinus thrombosis.

With regard to the extract, Dr. Dwyer said the main point is to give it often enough and to follow up the case. Local applications have no effect whatever, excepting on the mind of the patient. The great point is whether to isolate these cases or not. He has attended hundreds of these cases with other doctors, all over the city, and was also on a very active service with Dr. McKernon at the Manhattan, and never yet had he had personally a case of erysipelas. His plan would be to use personal precautions with the nurses in the wards, for it is possible that the infection might be carried on the hands of the nurses.

He has never seen any infection excepting with the streptococcus hemolyticus, and the erysipelas streptococcus belongs to that group. If the patient is left in the wards, and the same nurse observes sterile precautions and washes her hands, the disease will not spread. It has been known to follow certain men year after year; it follows their services, whereas in other services it never enters. The subject was a very timely one, for very little is known about it—the cases are shifted to the city hospitals and then lost sight of by the operator in attendance.

Dr. Hays asked if Dr. Dwyer thought the infection came from the mastoid wound.

Dr. Dwyer replied that in his opinion the infection in the mastoid is the infection in the erysipelas; the infection gets into the cellular tissue. Personally, he accounts for the length of time that elapses before the erysipelas develops by remembering that we have three clinically objective forms of erysipelas—cutaneous, cellulocutaneous, and cellulitis. The cellulitis, or deep form, may go on for some days before the condition comes to the surface. The cellulitis form does not usually go on to pus formation, and hence he believes it very poor surgery to make multiple incisions in these cases. An interesting point in regard to leucocyte extract is its use in infants. He had been told by leading pediatricians that the death rate in infants under one year is fully one hundred per cent. In his series, reported some years ago, the mortality was only thirty-three and one-third per cent.

Dr. Danziger asked if Dr. Hays had found that erysipelas is more virulent at certain times of the year. He himself has seen more erysipelas cases in the spring and early summer than at other times. Is that a coincidence?

Speaking of the therapeutic value of the blood of patients recovered from erysipelas, it would be interesting to know how soon he uses the blood, for we know that one attack of erysipelas does not make the patient immune. We know that he can have another attack, as in diphtheria.

Dr. Danziger also asked if Dr. Hays had seen the symptom described by Ruttin, "nystagmus toward the affected side," which, other symptoms being missing, make him suspect erysipelas. He himself has seen the early involvement of a lymphatic gland in the region of the submaxillary gland. Concerning the question whether the infection comes directly from the mastoid infection or is extraneous, it would seem that we have to differentiate as to the time when the infection occurs. If it occurs early in the infection, probably Dr. Dwyer is right; but cases have been seen where the wound had practically healed, and erysipelas suddenly developed.

Dr. Friesner, referring to the matter of pulse rate, said that while it is true that with erysipelas there is not usually a particularly rapid pulse, yet in Dr. Glogau's sinus case the pulse rate averaged ninety up to the time of operation. He himself has seen a number of cases of sinus thrombosis with a comparatively slow pulse—whether or not that meant increased intracranial pressure, due to venous stasis, he did not know; but too much stress should not be placed upon that point as a means of differential diagnosis. It is not at all infrequent for an erysipelatous patch to appear on the side opposite to the operated ear. It would seem that if the erysipelas were due to infection from the hair, it would always occur on the same side. Whether it be due to the organism in the mastoid wound or to an extraneous organism brought to the unhealed wound, is beside the question. It would seem that the cut or the granulated surface was the place of entry of the infection, the transmission being through the lymph channels. It has been often shown that hair may be dropped into culture medium and yet the medium remain sterile.

Dr. Blackwell said that practically all the cases of erysipelas occurring at the New York Eye and Ear Infirmary are transferred to Bellevue and treated there. He had formerly been house surgeon at Bellevue, and the number of remedies that had been tried there is legion; nearly all have their enthusiastic advocates. While he was on service there they clinically recognized two classes—of the head, and of the body. The large majority of the cases were confined to the head. If it stopped at that point, the patient recovered; but if it went below the collar line and below the chest line, it did not stop until it extended all over the body, and the patient was very sick. Fifteen years ago it was a matter of common observation that a large number of the mastoid cases that came in had erysipelas, and a point that struck all the men that had to treat these cases was the fact of the very great pressure caused by the tight packing of gauze in the mastoid wounds. At Bellevue, where all kinds of infective conditions are handled, the men are not accustomed to packing the wounds so tightly, and many of them attributed the frequent occurrence of erysipelas with mastoid to this tight packing. At one time it was the custom of many surgeons to lean on the packing and make it as tight as possible. and at that time the number of erysipelatous mastoids was much greater than now. In his opinion, one cause of the lessened number of erysipelas cases is the improved technic and the tendency to pack the mastoids more lightly.

As for the treatment, the method which had given the best results had been the administration of whiskey and saline solutions—cold applications of saline solution makes the patient more comfortable, and the whiskey stimulates him.

Dr. Seymour Oppenheimer said that in noting erysipelas in connection with mastoid operations, he had been impressed by the association of suppurative conditions in the accessory nasal sinuses. A few years ago he had made that statement, and at that time he did not recall having seen a single case of erysipelas without some suppurative condition in the nose. He is not yet prepared to state what relationship may exist between the two, but there is food for thought in the suggestion. It is quite possible that there may be an abrasion on the lip or cheek which becomes infected, and this infection is subsequently carried through the lymph channels to the aural wound. One point which should be considered in this connection is the development of what looks like an erysipelatous process, and which is a dermatitis due to iodoform. Dr. Oppenheimer said he has seen two cases where the local cutaneous lesion resembled erysipelas, and there were systemic manifestations as well. Both patients started off with severe chills, sharp temperature rises, and evidences of a hemorrhagic nephritis. In the first case it never occurred to him that it was iodoform poisoning. The process gradually subsided;

whether it was modified by the treatment could not be stated positively. This particular individual, it was discovered, was extremely sensitive to almost every drug. A slight quantity of xeroform dusting powder, employed when the wound was almost healed, produced the above mentioned local and systemic symptoms for the second time.

It is very curious to note a drug can produce symptoms so analogous to a bacterial infection.

DR. OPPENHEIMER said that he had had some experience with Hiss' leucocyte extract, and believed that it had some influence in reducing the temperature. Of local applications, alcohol was his preference, and he believed it to be of value in limiting the progression and severity of the local lesion. The ichthyol treatment of old is most disagreeable, and does not have much influence, and makes a very dirty mess. He had seen three cases develop erysipelas where the sigmoid sinus had been injured during the operation. No untoward symptoms, however, arose from this source. He also used defibrinated blood taken from another patient. Also the serum of the individual patient, but is not prepared as yet to state whether the influence of this line of treatment is of value.

DR. BRAUN said that he had seen a very unusual onset in two cases. Ordinarily one expects to see the erysipelatous rash appear within twenty-four or forty-eight hours after the rise of temperature, but in one of these cases the temperature rose and remained high for five days, and in the other it was six days before the erysipelas appeared. In the latter case he had almost made up his mind to explore the sinus, when the rash appeared.

DR. HASKIN said that some years ago he had read a paper before the section on the Hiss extract, and expressed his belief in it. He is not, however, a believer in the extract prepared by Squibb. It is made from the serum of a horse. It is a clear solution, and has not the appearance of the Hiss extract; he has used it two or three times, and the results were not satisfactory. The Hiss leucocyte extract is prepared from the pleural cavity of rabbits, and gives good results. Hiss, in his monograph on the leucocyte extract, states very distinctly that unless the leucocyte bodies were themselves injected into the tissue he got no results. Making a clear extract of the bodies of the leucocytes did not bring results; these were only obtained by injecting the triturated bodies of the leucocytes. Then you get apparently the building up of the leucocytes themselves, which gives increased resistance.

Dr. Haskin said he did not know whether or not the leucocytes are in the Squibb extract; it is a clear solution and does not resemble the extract prepared by Hiss. It seems a pity that they cannot follow the procedure which was followed by one of the greatest men we have ever had in this country or any other. Hiss had tried several animals, but could not get from the dog or other animals the results obtained from rabbits. It is greatly to be regretted that the Hiss extract seems to be a thing of the past. With the high cost of living, etc., the price of rabbits had gone up so that we cannot get the material. Dr. Haskin said he was glad to hear that Dr. Hays had obtained results from this extract. Perhaps he would himself try it again, but he certainly would not waste time with it in extreme cases.

Dr. Hays, closing the discussion, replying to Dr. Danziger, said that the greater prevalence of erysipelas during the spring and summer was probably due to the fact that it is then we have more mastoid cases. He had never observed the matter of nystagmus with this condition.

As regards the question of using the citrated blood, he regretted that Dr. Wright had not been able to be present, as he claimed to be getting some very good results. In most of the cases that occurred at Bellevue the patients had given positive Wassermann reactions, and it had not been possible to get much blood.

Dr. Blackwell had spoken of the tight packing of the ears in mastoid cases. Dr. Hays said that he himself never packs the mastoid, so that in none of these cases could the erysipelas be ascribed to that.

Dr. Oppenheimer had spoken of suppuration of the accessory sinuses. In the first case of erysipelas Dr. Hays had treated nine years ago, the patient had had a terrific sinus trouble after the erysipelas disappeared.

Dr. Hays said that he was surprised at Dr. Haskin's experience with the Squibb extract that had been shown tonight. Dr. Zinsser had advised him to go to Squibb for the extract, and he had naturally inferred that it was a good preparation; his results had certainly been very gratifying. In the original work he had done at the laboratory some years ago he had used rabbits for this purpose.

In response to a query as to whether any of these cases had had atrophic rhinitis, Dr. Hays replied that some of them had complained of dryness of the nose and throat, but that had naturally been ascribed to the fever.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of May 11, 1917.

Plastic Operation for Restoration of the Auricle, Following Injury From an Explosion.

Dr. Harmon Smith: Patient, aged thirty-five years, married; no previous history bearing upon the present condition. On April 28, 1915, he, together with three other councilmen of South Bound Brook, were called upon to investigate a sewer tank in the Raritan river. While in the tank there was an explosion of sewer gas, the cause of which was unknown. Two of the party were instantly killed by the explosion, a third was permanently injured, and the fourth, the patient whom I present, lay between life and death in the Monmouth Memorial Hospital for a period of more than five weeks.

He first came under my observation in February, 1917, nearly two years after the accident. Examination revealed the destruction of the upper part of the helix of the right ear, a union of the lobe with the tip of the mastoid and neck behind and below the lobe in one large keloid mass. The entire ear was held back by adhesions to the side of the head, while in front of the ear and on the neck were extensive keloids. The left ear was not so extensively burned, although the perimeter of the helix was destroyed on its superior tip for a depth of a quarter-inch. The left upper eyelid was also involved, so that there is an eversion and foreshortening of the lid. Under ether anesthesia, in February last, I undertook to first make him a new lobe, by cutting out some of the scar tissue below the ear and underlapping it to form a new lobe. Secondly, I made a curvilinear incision one and one-quarter inches back of the ear, and elevated the tissues down to the periosteum, which permitted the ear to be advanced forward. Thirdly, I took a flap from the healthy tissue back in the scalp, posterior to the burned area behind the ear, and leaving the base fixed superiorly, carried it over to the destroyed helix and fastened it there with horse-hair sutures, having first prepared the area for transplanting. This I left in place for six weeks before detaching from its base. All the operative procedures were successful and the patient made an uncomplicated recovery, except that during the time of healing there was a slight frost bite of a part of the transfixed tissue, which at one time threatened the favorable outcome of the operation.

I likewise removed a large keloid from the neck, about an inch long and one-quarter inch wide.

I present this case at the present moment to demonstrate in a measure, by showing the uncorrected side, the difficulties to be encountered when dealing with keloids and tissues which have had their vitality lessened by burns.

The patient also had his left hand severely burned, almost necessitating amputation, and at the time of operation I drew out what remained of the nails on his fingers. I attempted to skin graft the back of his hand, but the bleeding was so profuse that it was impossible to get a graft to take. I expect at the next operation to pursue about the same course, with regard to correcting the deformity of the left side.

DISCUSSION.

DR. PHILLIPS congratulated Dr. Smith on the remarkably satisfactory result achieved. In his own wide hospital experience he did not recall any case where more or less deformity had been so satisfactorily overcome. Dr. Smith had shown much mechanical ingenuity as well as skill in carrying out his ideas.

DR. QUINLAN said that he wished to second what Dr. Phillips had said. The result was certainly marvelous, and it was a pity that photographs had not been taken before and after the operation. The cosmetic effect was certainly most acceptable, and the profession, as well as the patient, was indebted to Dr. Smith for the time and skill in bringing about such a result. One seldom sees these external conditions corrected in such a satisfactory manner. Dr. Smith had more than once showed equally gratifying results by his ingenuity in paraffin injections.

DR. HASKIN said he had read that X-rays have been employed successfully on very extensive keloids, and asked if Dr. Smith had tried that treatment in this case.

DR. SMITH replied in the negative.

Chronic Otitis Media With Cholesteatoma—Unhealed Mastoid Wound—Cured by the Modified Radical Operation.

DR. JOSEPH H. O'CONNELL: The patient, twenty-five years of age, was admitted to the Post-Graduate Hospital on August 8, 1916. About a year previously a Schwartze operation had been performed. The posterior wound healed in due course of time, but the middle ear continued to discharge more or less freely. Later, the patient began to have pain, and the cicatrix broke down, leaving a sinus which existed when she presented herself for treatment. She complained of more or less pain and of having frequent attacks of vertigo. Examination showed that the lower portion of the brain was in a fairly normal condition. There was a small peripheral perforation in the posterior superior quadrant. When a bent probe was inserted into the sinus and down to the aditus, foul smelling cholesteatomatous matter could be easily forced through the opening in the sinus. The patient's hearing was very poor. Unfortunately, the record made of the hearing previous to operation was misplaced or lost.

On account of the good condition of the drum membrane, and feeling that the ossicles were not necrosed and that most of the trouble was in the antrum and aditus, it was decided to do a modified radical operation. The incision was made through the cicatrix, and the cartilaginous canal was separated from the bony wall. The antrum was enlarged, and the posterior bony wall was taken down. The field was enlarged at the expense of the pillars and the posterior bony wall lowered to the facial ridge. The external wall of the attic was carefully removed and all débris cleared out from the antrum and aditus. Care was taken not to disturb the ossicles and drum membrane. The operation was concluded in the ordinary way by suturing the posterior wound and firmly packing the antrum and aditus through the enlarged meatus. The patient recovered with a perfectly dry ear. She now feels well, has no more attacks of vertigo, and says that her hearing has been greatly benefited.

Dr. O'Connell said that he claimed nothing new in this case, but presented it simply hoping to stimulate otologists to further effort in this line. Dr. Hugh Blackwell, in reporting his results in a series of cases in which he had done the modified radical operation, concludes that this operation has the advantage over the ordinary radical in that the hearing is more likely to be improved. This case seems to bear out his claim, and this would seem to be the operation par excellence in selected cases.

DISCUSSION.

Dr. Phillips said that all must admit that sporadic cases of this type get well with this method of operation, but this section in otology should be very cautous in giving approval as to what may be expected in results from the operation if universally employed. Dr. O'Connell had wisely qualified his commendation by emphasizing the value of the work in a selected case. The operation should only be performed on suitable cases, and they are few and far between.

Dr. C. H. Smith said he had seen this case in the Post-Graduate clinic, and it seemed like a case that one would select for the radical mastoid operation. The discharge was very offensive, and the patient complained of vertigo, and he personally had felt very pessimistic about Dr. O'Connell's operation, and had been very much surprised at the excellent result obtained.

Dr. Guttman asked what had been found at the operation. Did the doctor find in the antrum and attic cholesteatomatous masses, pus, bone affected? Were the ossicles normal, and why did he stop there and not enter the tympanic cavity?

Dr. Voislawsky asked just where was the perforation in the tympanum.

Dr. Haskin said that the Heath operation had been very carefully studied by such men as Mosher, who followed up a series of cases, and the ultimate result was not found to be favorable. Dr. Haskin said he was afraid that later Dr. O'Connell would find, as Dr. Phillips had suggested, that the cholesteatoma would recur. That had been his own experience in two or three cases. They healed very promptly, but a nucleus remained and there was a reformation of the cholesteatoma.

Dr. Friesner said that the operation performed by Dr. O'Connell was not the Heath operation. Dr. O'Connell took out the attic wall and opened up practically all the spaces in the mastoid and middle ear which are subject to the formation of cholesteatomatous masses—that is, practically all, for in one case recently operated upon he had seen cholesteatoma that was not in the antrum, aditus nor tympanum, but in the hypotympanum.

As he understood, Dr. O'Connell opens the antrum, works out the aditus, takes out the bridge, and removes the outer attic wall that corresponds with the roof of the bony external auditory canal, leaving the incus and the head of the malleus exposed. The operation was not at all the same as the Heath.

Dr. Phillips said that he could show a series of cases of chronic suppuration of the middle ear in which he operated, doing what he called the intratympanic operation, where he believed that the disease was largely located, or centered in the attic and the outer attic wall, with indication of disease of the ossicles, removing not only the drum membrane, but the outer attic wall. Most of these cases occurred in private patients who refused to have the radical mastoid performed, and the results obtained were perfect; but he had had other cases in which the results were not good.

Dr. O'Connell, replying to Dr. Voislawsky, said that the perforation was in the posterior superior quadrant—Shrapnell's membrane. The cholesteatoma seemed to be in the antrum and aditus, and the reason he did not touch the tympanum was that from its appearance he believed it to be in good shape. As in certain cases the desired result is obtained by the Schwartze operation, in others by the removal of tonsils and adenoids, and in still others by the Yankauer operation, so in this class of cases he feels that the modified radical is better than the Stacke operation.

Dr. O'Connell said he did not claim that it was the Heath operation. Dr. Blackwell has done similar operations, and on comparing the methods they were found to be practically the same. It is an operation that is suitable for those cases in which the tympanum is not involved and the ossicles are in good shape.

Replying to an inquiry from Dr. Phillips as to the hearing results, Dr. O'Connell said that unfortunately the record that had been made of the hearing was lost, and he had only the patient's word for the result; but she claims that her hearing is better than before the operation. She now hears conversation at twelve to fifteen feet, and whisper at three feet. By leaving the drum membrane undisturbed, the result was probably better than if the eustachian tube had been curetted out. Where it is curetted out it is more likely to break down than if you leave a portion of the drum membrane.

Dr. Phillips said he was sure that the hearing would not have been just as good with the radical operation. He had one young patient who had suffered from otitis media suppurativa chronica nearly all her life. A radical mastoid was performed, and through some error the inner packing remained in the ear for nearly three months before it was removed; the ear was discharging all that time, yet the final result was perfect healing, and she could hear the acoumeter at twenty feet.

Acute Mastoiditis Followed by General Septicemia.

Dr. Wendell C. Phillips: C. H., aged twenty-two years, single. About Christmas time patient began having severe pain in left ear. In two or three days the ear started to discharge profusely, but, despite the discharge, the pain gradually increased in severity. From the first he had fever and great tenderness to pressure over the mastoid area. His condition became constantly worse, and at the time of his admission to the hospital, January 17, 1917, he was in great pain and at times irrational.

January 17th, simple mastoid operation. There was a large perisinus abscess. Following operation the temperature rose to 106°. (See temperature chart.) Culture from mastoid, streptococcus hemolyticus.

Blood count, January 17th: Leucocytes, 31,200; polymorphonuclears, 81 per cent.

Blood culture, reported January 20th: Positive streptococcus hemolyticus. Spinal fluid, January 26th: White cells, 21 per cubic centimeter. Globulin negative. Culture negative. Wassermann negative.

On January 22nd, sinus and juguljar operation. No clot was found. Two days after the second operation small petechial spots over the chest, and large hemorrhagic blebs formed in the skin on the hands and feet. Culture from these blebs showed staphylococcus. Blowing murmur at the base of heart. January 30th, death from sepsis.

Clinical diagnosis: Acute mastoiditis, followed by general septicemia.

AUTOPSY REPORT.

General Description.—The body is that of a fairly well developed, rather emaciated man. There is absence of body heat and rigor mortis throughout. The skin is yellowish to bluish white, with numerous small petechial hemorrhages distributed over it, especially numerous over the trunk. On the hands and feet, and occasionally elsewhere, are large blebs, some of which are hemorrhagic. The skin is loose over these areas, and upon removal small petechial spots similar to those described are present.

The head is swathed in bandages, with large pads over the right ear, and on removal the right side of the head is shaved, and there is a recent surgical incision in the mastoid region which is packed with iodoform gauze. There is also a second incision extending at right angles to this for a distance of three centimeters toward the occiput. Three centimeters below the first incision is a third one, extending for a distance of six centimeters along the anterior border of the sternocleidomastoid muscle. On removal of the dressings there is exposed a large cavity, which extends into the middle ear, and shows an entire removal of the mastoid cells with dissection into the lateral sinuses and downward, communicating with the external jugular. The dressings are blood stained, moist, and have no purulent exudate. The left ear has a sanguinous blood tinged discharge running from it.

The pupils are dilated, round, and about equal in size. The teeth are in bad condition, with abundance of sordes, and the mouth has some old and recent blood clot in it. The chest is long and narrow, with intercostal angle less than a right angle. The abdomen is scaphoid. Practically no panniculus. The muscles are deep red in color and show a moderate development.

Main Incision.—The peritoneum is moist, but there is no increase in fluid. The intestines are bluish brown in color and distended with gas. There are a few hemorrhages into the intestinal wall. The bladder contains a small quantity of urine, but is below the pubis. The appendix is negative. The liver in the right nipple line is two centimeters below the costal border and four centimeters below the ensiform. The spleen is behind the ribs, but definitely enlarged. The diaphragm on the right and left extends to the fifth rib. The sternum is negative.

Thoracic Cavity.—The left lung is free, and there is no free fluid. The right lung is extensively bound down at the apex and over the base with old fibrous adhesions. There is a moderate quantity of pericardial fluid, which is clear and straw colored; volume estimated at forty cubic centimeters.

Heart is about the size of a fist, is bluish red in color and has numerous small bright red spots, in size from a pin point to two millimeters in diameter, scattered over the surface. The consistency is rather soft and flabby. The tricuspid valve admits three fingers, the flaps are negative. The pulmonary orifice admits thumb with ease, and flaps are negative. The mitral valve admits two fingers. The valve shows several thickened plaques, and in one point there is definite ulceration, with erosion completely through the valve—the hole measuring approximately three millimeters in diameter. The aorta admits the thumb with ease, and valves are negative.

Skull cap is rather thick, otherwise negative. The dura is firmly adherent to the arachnoid along the longitudinal sinus. On removal of the dura the brain has a wet edematous appearance, with marked injection of the blood vessels. In the right frontal region there is an area of hemorrhage which appears to be hemorrhage by diapedesis. The sinus was opened from the point at which it was incised at operation to the torcular heropholii. A small chicken fat clot was present, but no free pus. The jugular bulb was also examined and no pus was found. An opening was made into the middle ear, and the anterior wall was considerably necrosed, allowing the passage of a probe, which entered the sheath of the carotid in the carotid groove. When this probe was passed, considerable free pus drained out into the cavity. The probe passed down into the sheath and the point came out to the skin surface just below the tip of the mastoid, showing a burrowing subdural abscess in this location. The portion of the dura covering the cerebrum and cerebellum at the point of operation showed no granulation areas or abscess formation.

Lungs.—Both lungs are motley dark bluish to light red and congested at the bases. The right lung is somewhat firm at the apex, and on section numerous small white areas are scattered throughout, giving the appearance of tubercles.

The spleen is considerably enlarged, motley bluish red in color, and at the pole it contains a firm area. On section this area is white in the center with a hemorrhage surrounding it (infarct).

The kidneys are smooth, capsule strips with ease. On section the cortex is pale and distinctly demarcated from the medulla. At one pole there is a white area surrounded by a zone of hemorrhage—this infarct being about one and one-half centimeters in diameter.

The liver is slate colored, smooth and rather firm in consistency. On section the edges turn back and the color is uniformly light yellowish red. The lobulations are indistinct.

The gastrointestinal tract is negative.

(The microscopic sections have not as yet been run through, but the findings will be made out as soon as examined. The pathologic diagnosis below is based on the gross findings.)

PATHOLOGIC DIAGNOSIS.

Nature and origin of fatal illness: Streptococcus otitis media with secondary mastoiditis and subdural abscess (dissecting downward and outward). Secondary: Endocarditis of mitral valve with thickening and ulceration of vegetations. Old and recent infarcts of spleen and kidneys. Extensive thrombosis of capillaries of the skin with production of petechiæ. Thrombosis of capillaries of pericardium and intestine with petechiæ. General streptococcus septicemia due to streptococcus hemolyticus.

Historic landmarks: Old apical tuberculosis of right lung, showing some evidence of recent activity.

DISCUSSION.

Dr. Friesner said that he had seen this case with Dr. Phillips at the Post-Graduate, and clinically it was a typical case of sinus thrombosis. The operative findings, to some extent, seemed to bear out that diagnosis, although there was no clot. It was difficult to reconcile the postmortem and the operative findings. Dr. Friesner said he had not had enough pathologic experience to know whether even a septic clot, in the bulb, for instance, might so disintegrate in six or seven days following a sinus operation that nothing could be found at postmortem. He did not know whether that was possible.

Dr. Guttman asked if the opening from which the pus came was in the middle cavity. While there may have been a septicemia present, it did not seem to be a typical sinus thrombosis temperature. If the pus broke through the inner surface of the tip and went down the neck, it may have been something similar to a Bezold mastoiditis.

Dr. Phillips said that he was not present at the postmortem, but the findings were that the anterior wall of the middle ear was necrosed and a probe passed down the carotid sheath gave vent to considerable free pus.

The primary operation was a very extensive one, the tip and everything possible was removed. Dr. Guttman was evidently laboring under a mistaken impression. It was not found at the sinus. It was the anterior wall of the bony wall cavity —the tympanic cavity.

Dr. Haskin said that Dr. Page had recently reported a case of hematogenous meningitis and cerebritis in which at autopsy no clotting was found in the sinuses.

Dr. Hubby said that the cardiac postmortem findings seemed sufficient to account for the temperature. It did not seem necessary to consider the sinus at all, so far as the explanation for the temperature was concerned. It would be interesting to know if there was any previous septic history to account for the old infarct of the spleen that was found at the autopsy.

Dr. Voislawsky told of a case to which he had not paid much attention at the time, so far as the postmortem findings were concerned. At the time of operation he had been obliged to resect almost to the torcular and remove the jugular; but at postmoretm no clot was found, nor anything remaining in the sinus. Dr. Phillips had spoken of "chicken fat clot." Dr. Voislawsky said he did not know what that was. Dr. Page had had a case in which nothing was found in the sinus—making three cases of this kind. This brought out a point that had never before been discussed in the section. What becomes of the clot? In our sinus operations a clot forms behind the packing, and yet here are three cases in which there was no evidence of a blood clot.

Dr. Friesner said that sepsis from an ear through the lateral sinus does not necessarily entail, in all cases, a thrombosis. The bacteria can go from an infected mastoid through the vein of the lateral sinus without any protection in the sinus at all—the usual protection in the sinus, the throwing out of the clot, need not occur. One of the points that might be emphasized was that given a badly infected ear and mastoid and a bacteremia, one should always consider—and seriously consider—the advisability of shutting out the venous stream from the ear into the general circulation, which means a sinus operation or exploration and ligation or excision of the jugular.

Dr. Seymour Oppenheimer said he did not think Dr. Friesner's viewpoint was correct—that one could have an infection of the blood stream without a thrombosis. One may have a parietal clot which is so small as to escape observation, as it may be washed out in the blood stream after incision of the sinus. In several cases that he had seen there was a positive blood culture and bacteremia, on the strength of which he had opened the sigmoid sinus, but failed to find the thrombosis; yet after that procedure a negative culture was obtained. He expressed the belief that in every case there is a thrombus, although it may be so small that it may not be recognized at the time.

Dr. Haskin asked how Dr. Oppenheimer accounted for failure to find it even at autopsy. Dr. Page's case had been very carefully examined and there was no clot.

Dr. Oppenheimer said that he did not know how to account for it, but simply stated the above as a clinical impression based on experience.

Dr. Haskin expressed the opinion that in these cases there is an infection that takes place in the system, but does not take place in the sinus. Why may the infection not be carried out through other channels? They may communicate with various little ducts going through the anterior wall. These cases are probably much more common than is recognized. He had been very fortunate in his death records, having had only three. One of these was a child who was brought into the hospital in a comatose condition. He operated, but without avail, and at the autopsy the whole carotid canal was filled with pus. One could not say where it went through the canal, but it seemed reasonable to suppose that it went through the middle ear itself. If one examines many bones, dehiscences will be found. Why should not an infection spread in this way as well as through a lateral sinus?

Dr. Phillips, closing the discussion, said that as far as the "chicken fat clot" was concerned, he could only say that that was the way the postmortem described it.

He had greatly enjoyed the discussion of this case. In many instances where he had operated on lateral sinus cases he had been disappointed in not finding any clot; in the majority of these cases the patients lived, but there was not any clot. In regard to this particular case, he did not believe that one otologist in fifty would have failed to operate upon it as a case of lateral sinus thrombosis. The patient died of the septic endocarditis.

Replying to Dr. Friesner's remarks, Dr. Phillips said that they had many times discussed this question together, but that he had come to the conclusion that this infection and bacteremia never went through the lateral sinus, but that it probably went into the circulation through the carotid canal.

Sinus Thrombosis Following Fracture of the Skull; Subsequent Acute Mastoiditis, Erysipelas—Operation, Recovery.

Dr. William Wesley Carter: The patient is a well developed man, forty-three years of age, a waiter by occupation. His family history has no bearing on his present condition. He denies specific infection. Is a moderate drinker. Two years ago he was struck over the head with a blackjack, but recovered entirely from the injury. On February 13th, while waiting for a trolley car, he was struck by a truck. He was brought to Gouverneur Hospital in an unconscious condition.

Examination.—Patient unconscious, skin over right eye swollen, and sclera of this eye ecchymotic. Pupils are unequal, the left being quite small and does not respond to light; the right is dilated. Colles' fracture of the right wrist. Profuse hemorrhage from right ear. A clinical diagnosis of fracture of the skull was made.

X-ray examination shows a linear fracture of the skull, right temporal region. The right frontal sinus is obscured, probably being filled with blood.

Three days after admission patient's normal mental condition became clear. Hemorrhage from the ear continues.

On February 18th, five days after admission, I was asked to see the case on account of the persistent hemorrhage from the ear. I advised irrigations with hot boric acid solution, to be followed by a solution of adrenalin dropped in the ear; also ice bag to the side of the head. Within twenty-four hours the hemorrhage ceased.

Patient's condition was apparently satisfactory until March 6th, twenty-one days after the injury, when he complained of very severe pain in his right ear, the ear from which the hemorrhage occurred. This pain extended down the side of his neck. The temperature, which had been normal since admission, began to rise and reached 105 degrees. The urine showed a large amount of albumin and some hyaline casts. Blood culture negative. Blood count: white cells, 11,500; polymorphonuclears, 76 per cent. There was no tenderness over the mastoid bone, but the tenderness over the upper two-thirds of the sternocleidomastoid was exquisite. The muscle was considerably swollen. The patient could not move his head, was in great pain, and begged piteously for relief.

Thinking of a probable sinus thrombosis, I decided to operate, though there were no classical symptoms of mastoiditis present.

The mastoid antrum and all of the mastoid cells, which were large and extensive, were found filled with clotted blood.

In view of the swelling and tenderness in the neck and along the course of the jugular vein, and the fact that the sinus showed almost black through its thin bony covering, I uncovered the sinus. It was dark blue in color, and on indenting it with a probe it did not resume its normal shape—evidently it was filled with a clot. I made an incision into it—there was no bleeding. I then uncovered about one inch of it, and with a pair of bayonet forceps I withdrew from the direction of the torcular a firm clot about three inches in length, which reached perhaps as far as the torcular. Free bleeding immediately occurred. The clot was composed in its outer half of fibrin, showing, I presume, that clotting had occurred slowly and had proceeded from within outwards. After plugging the distal end of the sinus I succeeded in removing a similar clot from the direction of the bulb. So firm was this clot and so loosely adherent to the sinus walls, that it showed when removed the curve of the sigmoid sinus. In my exploration of the sinus and the adjacent bone I could discover no fracture. I then exposed the jugular vein, and finding that there was no clot below the entrance of the facial vein, I tied it. Cultures made from the clots removed from the sinus proved them to be sterile.

Shortly after the operation the temperature went to 106°, but in three days it fell by lysis to normal. The patient was relieved of his excruciating pain in the neck and became rapidly convalescent. At times he showed signs of mental aberration, and he complained of headache. I attributed these symptoms to disturbed cerebral circulation.

On March 23rd, two weeks after operation, the patient complained of pain in the left ear. Paracentesis was followed by a purulent discharge (streptococcus). Two days later tenderness and swelling developed over the mastoid. Temperature, 105°. Mastoid operation was performed on March 28th. The mastoid cells were all filled with pus. Culture showed a streptococcus infection.

Two days after the operation erysipelas developed around the mastoid wound; this spread over the side of his head. Temperature, 105°. Erysipelas was under control in five days, and since this time the patient has been slowly recovering, and he expects to leave the hospital in a few days.

Aside from the interesting features of this case, I feel that it is important to direct your attention to this class of injuries, very dangerous in their nature, sometimes extremely obscure in their symptomatology, and which may, as in the present instance, involve those parts in which we are especially interested.

DISCUSSION.

Dr. Hubby said that it was remarkable how seldom one sees a fracture of the temporal bone become infected. Many such fractures were brought to the Harlem Hospital, but in three years he had seen only one become infected, and that occurred about two weeks ago. The mastoid was operated upon, and the patient had just gone home. The convalescence pursued the ordinary course following the simple mastoid operation for acute mastoiditis.

Dr. Guttman asked where the fracture was. According to the X-ray plate the fracture seems to go through the frontal bone. That would explain the blood in the frontal sinus, and the fact that the other sinuses were also implicated. It would be interesting to know how Dr. Carter explains the infection of the frontal sinus infecting the mastoid. It is generally accepted that in fracture of the base of the skull with blood in the middle ear it is very unwise to irrigate the middle ear, for such a procedure is apt to produce an infection. The doctors must never expect to see a thrombus of the lateral sinus reproduced on an X-ray picture.

Dr. Dwyer said he believes that the vast majority of clots are nonseptic to start with—the clot is formed before the infection. In bacteriology it is well known that if fresh plates are used with the blood poured on at the ordinary temperature of forty or forty-five degrees, it will inhibit the growth of the organisms; therefore, at the Manhattan they never use these fresh plates. Time and again they have used negative blood plates that had stood only a few days. If the blood is mixed at fifty or sixty degrees to destroy the normal immune bodies present in normal serum, immediate growth is obtained.

We first have the clot in sinus thrombosis; we have a practically sterile clot; if the infection continues, we get liquefaction of the blood and infection. The large majority of clots are sterile. If enough bacteria get in and infection occurs and operation is not performed, all the pathologic changes follow —coagulation of the blood, liquefaction of the clot, and then infection of the clot and blood stream; but the big majority of the clots are sterile and remain so.

Dr. Haskin said he had never seen such a case—with the infection extending over the whole side of the head.

Dr. Dwyer said he has not seen any cases, but at the college there was a collection of pathologic specimens, and it was his impression that it was very rare for the Welch bacillus to go as far as in the case which Dr. Carter showed and not go further. The point made by Dr. Carter that the anaerobic bacilli are carried by the blood stream and use up oxygen is not accurate, because it is not in a free state. The anaerobe has to take its oxygen from organized material or carbohydrates. The gas infections in the French Army, up to the time they started using modified vaccines, have been very fatal. Was Dr. Carter aware that it has been identified with the Welch bacillus? It has been very carefully studied and can now be easily identified.

DR. CARTER, replying to Dr. Guttman's inquiry in regard to the fracture, said that the fracture shown in the X-ray plate extends in the direction of the frontal sinus, and unquestionably goes into the sinus. The patient had bleeding from this side of the nose also, the blood coming, no doubt, from the frontal sinus. The fracture that caused the clot in the lateral sinus was at the base of the skull. An unusually large number of plates had been made in the effort to find this fracture, but it could not be identified on account of the numerous osseous planes at the base of the skull. Dr. Erdmann and other surgeons saw the case, and they all agreed that there was probably a fracture at the base of the skull.

In regard to irrigating the ear, the blood clotted in the meatus, and that was why irrigation was employed; but it was carefully done, there was no undue pressure, and little or no danger of carrying infection to the meninges. The expedient of dropping adrenalin in the ear and applying ice bags to the side of the head evidently stopped the bleeding, which was profuse and had persisted for five or six days. It occurred to Dr. Carter that the injury to the sinus might have been caused by contre-coup. This could have caused the thrombosis without there necessarily being a fracture at the base of the skull; this, however, was merely a surmise.

In regard to the clot itself, a very good pathologist had made two or three cultures and they were absolutely sterile. He also made a culture from the mastoid cells, which were all filled with clotted blood, and they were all sterile.

Dr. Carter said that there was one point upon which he would be glad to be enlightened. In this instance a sterile clot was removed from the sinus, the patient's temperature dropped from 106° to normal, and he became convalescent. Now, why should the temperature come down and the case continue to progress favorably simply by substituting the clot which would form after plugging the sinus for the original clot which was due to the fracture?

In regard to the second case, that of gas bacillus infection of the mastoid, the pathologist was positive that he had identified the organism as the Welch bacillus. Dr. Carter said that personally he knew nothing about this, for he was not familiar with the bacteriology of the disease. His own idea in regard to the blood stream carrying the bacillus was that in cases which prove fatal it does not produce gas in the body until after death, and then there is a very rapid production of gas which makes the body swell. It is very unusual for the gas bacillus to cause symptoms so mild as in this instance. The patient, however, was only thirty-two years of age, was unusually strong, vigorous and healthy, and had good habits and a good constitution. Moreover, the distribution of the toxins through the body might have been limited somewhat, due to the locality which was infected.

Dr. Carter said that he had reviewed all the English literature bearing on this subject, and in only one instance did he find reference to the fact that gas bacillus might be found in the mastoid. There was not a report of a single case.

DR. HASKIN said that he thought Dr. Quinlan had reported a case after the Spanish-American war.

Cerebellar Abscess Complicating Acute Mastoiditis.

DR. ISIDORE FRIESNER: George C., seventeen years old, came to Dr. Phillips' clinic at the Manhattan Eye, Ear and Throat Hospital with an acute otitis media and mastoiditis (right) on September 19, 1916. A myringotomy was done under local anesthesia. Two days later he returned to the clinic. There was scant purulent discharge without odor coming through a good sized myringotomy opening in the posterior inferior part of the drum. There was no perforation elsewhere in the drum, although, on questioning the boy, he stated that the ear had discharged several times before the present attack. He had torticollis, held his neck rigid and his head inclined toward the side of the diseased ear. Pressure on the right sternocleidomastoid muscle, several inches below the tip of the mastoid, caused great pain. The mastoid was tender, and there was slight edema of the overlying parts at the tip. He looked sick. Temperature, 102°; pulse, 100; respirations, 20.

He was admitted to the hospital and a simple mastoid operation was done at once. The mastoid was pneumatic and very extensive. The cells were filled with pus and granulations. The cells continued for quite a distance internal to the digastric groove toward the foramen magnum. Neither dura nor sinus was exposed.

The following day the temperature had dropped to 98.8° and the pulse to 90. He whined considerable, but made no definite complaint. He had no headache, ate fairly well, and his bowels moved normally. For the next four days the temperature ranged between 98.5° and 99.5°, pulse from 76 to 100. He slept well. A full dressing on the fourth day disclosed nothing abnormal in the bony wound.

Early on the morning of the fifth day he vomited, complained of headache and became very drowsy. Temperature, 99°; pulse, 40. Involuntary stool and urination.

Cerebrospinal fluid was not under tension, looked clear, contained a few pus cells but no bacteria, either on smear or culture. The blood count showed leucocytes, 26,000; polymorphonuclears, 92 per cent.

The pupils were equal and reacted to light. The eye grounds were negative. There was neither nystagmus nor conjugate deviation. The neck was slightly rigid. There was no Kernig. The abdominal reflexes were lost. Knee jerks increased—about equal. Bilateral Babinski; this was definite, but there was no Oppenheim, Gordon or ankle clonus. On account of the mental condition, pointing tests were impossible.

Exploratory operation (with very little anesthesia). The dura of the middle fossa was exposed first. It was normal. Then the posterior fossa was exposed, taking out the angle between the middle and posterior fossæ. The sinus and cerebellar dura also appeared normal. Somewhere between ten and fifteen minutes were consumed in exposing the dura. At this time respiration suddenly ceased. The cerebellar dura in Trautmann's triangle was hastily opened and a brain knife introduced into the cerebellum in a downward and backward direction. No pus. A pair of bayonette forceps were then introduced, following the same direction which the knife had taken. The forceps entered the brain about one inch. A little over an ounce of thick, yellow, nonodorous pus welled rapidly out of the cerebellum. Stimulation, artificial respiration, pulmotor were all of no avail, and twenty minutes after respiration had ceased heart action also ceased.

The dura was examined over the lower portion of the cerebellum and over the anterior part as far inward as the internal auditory meatus. Macroscopically it was normal; there were no granulations and no exudate. The sinus, too, was normal.

Culture from the mastoid at the first operation showed short chain streptococci. Culture from the pus from the cerebellar abscess recovered the same organism.

DISCUSSION.

DR. HERZOG asked whether Dr. Friesner went through the original mastoid wound.

DR. FRIESNER replied that he went through the original wound.

DR. HELLER said that Dr. Friesner reported the case as one of acute mastoiditis, and yet said that the boy gave a history of having had discharges from the ear previously. Was it not possible that it was not an acute case but a recrudescence of an old condition?

DR. FRIESNER said that the boy gave a history of having had attacks of ear trouble with discharge before, but when he came to the clinic he had an intact drum. It was red and bulging; particularly in the posterior quadrant. A myringotomy was done under local anesthesia. He returned in forty-eight hours, and was again questioned; and he said that he had not had any discharge from that ear for along while, although it had previously discharged, but that each time he had had severe pain in the ear before it discharged. Frequently, where there is a permanent perforation, the ear discharges without pain. Here there was pain for two weeks, but no discharge. Not only did the history suggest an acute attack, but it was impossible to find any perforation except the one made for the relief of this attack. Moreover, the findings in the mastoid itself suggested an acute mastoiditis. It was a very pneumatic mastoid, with large cells which were full of pus and granulations.

Dr. Guttman said he believed it is an accepted theory that abscesses of the brain are formed by continuity—the pus extending from the mastoid to the brain. Considering that this was an actue case, did Dr. Friesner consider that the infection came to the cerebellum by infection from the middle ear in such a short time?

Dr. Friesner said that while it is true that in many instances brain abscesses are formed by continuity, it was by no means true that all brain abscesses are so formed. He had seen a number of brain abscesses with normal dura and normal brain tissue between the abscess cavity and the dura.

Dr. O'Connell, in reply to an inquiry as to the possibility of the patient being a hemophiliac, said that so far as he knew there was no such condition. The patient had recently had a submucous operation performed, and there was no trouble. Both drums were incised at the same time, and there was no trouble in the left ear, while from the right it was as stated. The patient was a healthy looking young man, only twenty-three years old.

Dr. Haskin said that it was not unusual to find the roof of the bulb in very excellent position to be punctured through the middle ear. In a number of specimens he had found the roof of the jugular bulb rise under the labyrinth and the promontory; and in many cases, if the bones are held up to the light, one can see dehiscences in that region, and in such cases the bulb might readily be entered if the incision were made below the foramen rotundum.

Otitic Meningitis.

Dr. Seymour Oppenheimer said that while there was nothing very unusual about the case, yet cases of this kind always form an interesting subject for discussion.

On April 14th he had been called to New Rochelle to see the patient, a man, fifty years of age, who had been suffering for several days with a slight suppurative discharge from the middle ear, spontaneous perforation having taken place with very few initial symptoms. During five days he had had a very slight head cold, with the highest temperature recorded being 100.5°. On the fourth day he was practically discharged by his physician, with instructions to report in a few days.

On the morning of the fifth day he had a chill, and in the afternoon a temperature of 104°, a double Kernig and a rigid neck. There was no tenderness on pressure over the mastoid. It was decided to operate, and the mastoid cortex was found to be very thick, which accounted for the fact that there was no external tenderness on palpation. During the operation a lumbar puncture was made, and the fluid was found under pressure and extremely turbid. While the patient was still on the table a smear was made from the fluid and examined under the microscope, and showed a large increase of the cell count, but morphologically nothing was found suggestive of bacteria. Dr. Oppenheimer said that he did not perform a cranial decompression operation, although he certainly would do one the next time he had a case of otitis disease with meningitic symptoms and turbid fluid. A number of white mice were injected intraperitoneally with the cerebrospinal fluid. In this way determination could be made quickly as to whether they were dealing with a bacterial or a nonbacterial meningitis.

On the following morning the patient's condition was somewhat improved, and all the cultures taken and the white mice inoculations failed to show any organisms, so that they were inclined to feel that the meningitis was not bacterial. In the afternoon, however, the patient's temperature rose rapidly and the evidences of meningitis became more marked. He was accordingly placed on the table again and a decompression operation was performed.

Dr. Oppenheimer said that this was the last time he would ever attempt a subdural drainage by this route, as it does not drain the subdural space but for a very short time, but he would in the future rely on decompression and lumbar puncture rather than subdural drainage. Some smears were made and morphologic evidence of some organism was found, but it could not be identified, although it was believed to be a streptococcus. More white mice were injected, and in six hours they showed the evidence of a streptococcus mucosus infection. . An interesting point in this case was that the operation was performed evidently just between the serous and the purulent stages of the meninigitis. Another point was the seriousness of all these cases of streptococcus mucosus infection. They may well be called "a thief in the night." This patient's otitic symptoms were very mild, yet he had this rapid flare-up, the development of meningitis, going from bad to worse and succumbed in a very short time.

Dr. Oppenheimer said that in this case he had withdrawn a quantity of blood from the vein of the arm of the patient, getting about ten cubic centimeters of serum from the blood and having this injected into the spinal canal after the spinal puncture. The serum of the blood contains the antibodies and forms the theoretical basis for this method of procedure. It had occurred to him that it might be feasible to keep track of these streptococcus mucosus cases—cases that have recovered from an acute mastoiditis or otitis—where this particular type of infection had been present, and utilize the antibodies of their blood for introduction into another patient, in the same manner as this plan of procedure has been used in cases of poliomyelitis.

DISCUSSION.

Dr. Heller said that as Dr. Oppenheimer mentioned he had operated upon the case between the serous and the purulent stages of meningitis, he would like, in this connection, to make a preliminary report on a case which had been admitted to Lebanon Hospital last week with a diagnosis of meningitis. The patient, a boy, was practically unconscious when he was brought to the hospital, and had a temperature of 105.8°. Lumbar puncture was performed and fluid under great pressure was removed—five hundred cells per cubic millimeter. The fluid was sterile. The medical men were convinced that it was meningitis and nothing else. He had Babinski and all the other signs. It was learned that he had been operated upon a year before at the Manhattan Eye, Ear and Throat Hospital for some ear trouble, and that there had been a discharge ever since. The boy was in a desperate condition, and while there was very little hope, yet it was thought best to operate immediately. It was found that a modified radical operation hal been performed. The posterior wall was gone, but the ossicles were still present. The tympanic cavity was opened and cholesteatoma was found. When the roof of the antrum was opened the dura bulged through the opening. This was incised and drained. The next morning the temperature was down to 101° and the boy was conscious. Then it rose to 102° on the third day and the boy had pneumonia and showed pneumococcic serum in the blood. He is still alive with an active pneumonia.

Dr. Haskin said that these cases are very puzzling, and then cited the case of a patient on whom he had recently done a mastoid operation. She had been operated upon seven years previously, but suffered constant pain, and it was finally decided to reopen the ear on account of this pain. Later, she started a temperature, and it continued at 105° to 107.2° for about fifteen days. She looked sick, and yet all the indications were against doing anything. She went from 25,000 leucocyte count to 8,200, and showed a very interesting blood series. The girl had absolutely no clinical symptoms; all the reflexes were negative; Wassermann, typhoid, and tuberculin complement fixation tests were also negative. The patient was very sick, and the family wanted him to operate, as did also the family physician. He refused, however, and they decided to take her to the Neurological Hospital, carrying her out with a temperature of 106°. It was only a few blocks to the hospital, and when she was admitted there her temperature was normal, and never again went over it. The only way to account for it was that she had had a basilar meningitis. She had a partial facial paralysis, appearing three weeks after operation, but they had hesitated to do a lumbar puncture—for Dr. Rae had done one under somewhat similar circumstances and later a decompression operation, but the patient had died, and he advised against lumbar puncture. When it came to the 20th of March and everyone thought she was going to die, a lumbar puncture was made to keep the record complete; twelve cubic centimeters were withdrawn and replaced with normal saline. The fluid was absolutely normal chemically, cryptologically, and bacteriologically. Whether that had anything to do with relieving the pressure no one will ever know, but the temperature dropped in a journey of a few blocks and never went up again.

Dr. Herzog asked if Dr. Oppenheimer had used Hiss' extract.

Dr. Seymour Oppenheimer replied that he had not used Hiss' extract intraspinally, but had injected urotropin. The last case so treated had had some very violent convulsions following the injections, and he believed the end was hastened. If his understanding of the benefit to be derived from urotropin was correct, it must be introduced or secreted through the cerebrospinal fluid. The profession unfortunately must admit its helpless position when confronted with such cases.

DR. HASKIN said that urotropin has to be acted upon by an acid to generate formalin, and he could not understand the rationale of introducing it into the spinal fluid.

Gas Bacillus Infection in a Chronic Mastoiditis With Cholesteatoma—Operation, Recovery.

DR. WILLIAM WESLEY CARTER: The patient, a large, powerfully developed Russian, thirty-two years of age, was admitted to my service in Gouverneur Hospital, March 16th. Family history is unimportant.

Aside from the ear condition leading up to his present illness, patient has always been strong and healthy, and says that he was never really sick before in his life. He denies venereal history, and says that he neither drinks nor smokes.

Following an attack of la grippe two years ago, he had an abscess in his right ear. At intervals since this time he has had pain and a discharge of pus from this ear. He has never received treatment for this condition. At times the pain in his ear has been very great, but it has always subsided after a profuse discharge of pus.

For three months he has had a bad cold in the head, and for the past month he has had almost constant pain in the right ear. Several days ago the discharge from his ear increased greatly. On the day before his admission to the hospital he had a chill, followed by fever and sweating. The tissues around the ear became swollen and painful, and this condition gradually extended over the whole side of his face and head.

Examination shows the following condition: General appearance of patient is that of a very sick man. Temperature, 102°; pulse, 90; respirations, 24. Right side of the face is paralyzed, right eye almost closed, entire right side of face, neck and head greatly swollen, and skin is dark red in color. The limit of the swelling is fairly distinct, but it has not the prominent line of demarcation characteristic of erysipelas;

then, too, the swelling is bulging in character, the most prominent point being over the mastoid antrum. There is a foul, purulent discharge from the right ear. No attempt was made to inspect the drum, as the canal was closed by the swelling. On account of the pain and swelling the patient can scarcely open his mouth wide enough to take nourishment. The entire swollen area is very painful to the touch. Firm pressure with the fingers gives a sense of fine crepitation, which is combined with that of fluctuation over the mastoid. Blood examination: White cells, 12,000; polymorphonuclears, 78 per cent.

Operation.—The usual incision for mastoiditis was made. As soon as the knife penetrated the deep layers of the skin a most unusual phenomenon was observed: the blood oozing from the wound appeared to be charged with gas which was escaping in fine bubbles. The incision was made fully an inch and a half deep, through the edematous emphysematous tissues before the periosteum was reached; as soon as this was incised there was an almost explosive discharge of foul smelling gas, followed by thin fetid greenish yellow pus. The odor of this discharge was said by all of those present to be identical with that of other cases of gas bacillus infections treated in the hospital.

Subperiosteal elevation of the tissues extended over the entire right side of the head.

There was a large perforation in the mastoid cortex at a point corresponding to the antrum. The entire mastoid process with the exception of the extreme tip was occupied by a cholesteatoma which extended also into the middle ear. The posterior bony wall of the auditory canal was completely destroyed.

The small vessels lying upon and entering the bone over the entire affected area presented a very unusual appearance—they were thrombosed and resembled fine black hairs.

The cholesteatoma and the tip cells, which were filled with pus, were thoroughly removed, and all overhanging bone was curetted away. Free counter openings for drainage were made through the scalp; one near the parietal eminence and one near the occipital protuberance. The wound was irrigated with hot normal saline solution and packed with iodoform gauze.

A culture made from the wound pus showed a mixed infection, including large numbers of giant rod-shaped gas bacilli. These bacilli are Gram positive, are about 5 µ. in length and about the thickness of anthrax bacilli. They correspond to the description of the Welch bacilli, which have been described by Nutthall, Flexner, and others.

Following the operation the patient showed almost immediate improvement. The temperature in two days dropped to normal; his recovery was uneventful, and he was discharged from the hospital on April 5th, nineteen days after admission. The wound has since healed entirely.

The gas bacillus is anaerobic; it cannot live in oxygenated, circulating blood. It would seem, therefore, that an old cholesteatomatous mastoid would offer ideal conditions for its propagation where once it has gained an entrance.

Interesting features of this case are:

1. The rarity of gas bacillus infections of the mastoid.

2. The unusual route through which the infection gained an entrance—namely, through the middle ear. The usual mode of entrance of this bacillus into the body is through an open wound or abrasion into which earth has been ground.

3. The prompt recovery following the operation: an unusual sequel to gas bacillus infections, for these cases usually succumb very quickly.

Profuse Hemorrhage From the Middle Ear Twenty-four Hours Following Incision of the Membrana Tympani.

Dr. Joseph H. O'Connell: On December 14, 1915, Abraham W., aged twenty-three years, was admitted to the Post-Graduate Hospital suffering from severe pain in both ears. Nine days previous to admission a submucous operation had been done. On the morning of his entrance to the hospital he was awakened by a sharp pain in the left ear; a few hours later pain lighted up in his right ear.

On admission his temperature was 101°; pulse, 100; respirations, 24. Examination of the ears revealed drums acutely inflamed and bulging. A double paracentesis was immediately done under gas anesthesia. The smear which was taken and later examined showed a streptococcus hemolyticus infection of both ears. The following morning the patient was very comfortable.

On the afternoon of the second day he began to bleed from the right ear. The bleeding started suddenly as he was lying quietly in bed. When I saw him blood was flowing freely and several towels were well saturated. The blood was dark in color, and a large clot was protruding from the meatus. No blood appeared in the pharynx. After thoroughly cleansing the canal I could distinctly see the blood oozing through the incision which had been made in the drum. As soon as it was wiped away it immediately reappeared. There was no pulsation. Slight pressure with cotton on an applicator was sufficient to stop the flow. A small strip of gauze was packed against the drum and this readily controlled the hemorrhage. On the following day the gauze strip was removed, and although the ear continued to discharge for some time after, there was no recurrence of the bleeding.

Profuse hemorrhage from the middle ear in adults following incision of the drum is not very common. It is more likely to occur in infants and very young children because the bulb, which may be high up, is not protected by firm bone, or a dehiscence in the bone may still persist.

In my case, the wall of the bulb may have been injured through a dehiscence during the paracentesis and later it ulcerated through. The only other source of profuse hemorrhage in this region would be from the lateral sinus. But to injure the sinus through the middle ear is not at all probable unless an anomalous condition exists.

NEW YORK OTOLOGICAL SOCIETY.

Meeting of November, 1916.

Neoplasm of the External Auditory Canal Treated by Radium.

Dr. Harris: About three years previously the patient was seen for the first time, when he complained of a slight discharge from his ear. At that time the condition was about as follows: The drum could be seen, there was a discharge in the canal, and he had superficially all the landmarks of chronic suppuration of the middle ear. The fork tests showed absolute destruction of the labyrinth. Dr. Harris could get no response whatever. The patient had an impression that he heard, but the speaker had never been able to discover that he had any hearing in the affected ear. In the canal there was a mass which suggested granulation tissue, which bled very easily when probed. Dr. Harris had the patient under observation for about one month, at which time the pain of which he complained rather increased, and he also had several spontaneous hemorrhages. At that time, a month after the beginning of treatment, an X-ray picture was taken of the mastoid, and interpreted by Dr. Law, who said there was a spot of disease and pus in the mastoid antrum. The patient was advised to have an operation performed at that time. This was three years ago in April. Dr. Harris went into the mastoid and, assisted by Dr. Haskin, found a healthy mastoid, but when they opened up into the canal and attempted to remove the mass which was there, they encountered such tremendous bleeding that, after getting out a small bit of the growth, they were compelled to desist. This was submitted to the pathologist at St. Luke's Hospital, who reported it, so far as he could determine from the small piece submitted, appeared to be a malignant growth. The case was kept under observation. There was no return of the growth whatever, and the patient went back to his profession, that of clergyman.

The next time Dr. Harris saw him, about one year later, he returned with no complaint about the ear, but paralysis of the left vocal cord, which was absolutely immovable, had developed. That condition was giving him distress because he could not speak. Six months later, in the spring, he presented himself with the complaint that his ear was again giving him trouble. At that time Dr. Harris found the canal completely filled by a new growth. This was giving him some pain, and what was evident on his presentation before the society was more in evidence then. There was a facial paralysis, involving the eyebrow and muscles of the face on the left side. Dr. Whiting saw the case in consultation, and the question was considered how thoroughly the tumor could be eradicated. Dr. Harris felt that before proceeding to this very radical procedure he would try something less radical.

Last spring the patient received eight applications of radium at weekly intervals, and four this fall, making altogether twelve applications. There had been a very noticeable diminution of the growth. It began in the canal, and had been entirely flush with the outer entrance of the ear. The symptoms disappeared. The patient had informed Dr. Harris the day previous, and he was satisfied he was getting decidedly more power over the facial muscles, and while there was as yet no movement of the vocal cord, there was without question movement of the left arytenoid cartilage. The patient had been able to pursue his occupation of clergyman in a very large church in town without losing a single day. He felt perfectly well, and had full weight and strength.

Dr. Harris said the members might ask the question why he did not confirm the diagnosis of the pathologist by another section, but he simply felt it unwise to take any chances of hastening the growth by taking out a sufficient section of the tumor to make a correct diagnosis. He thought the case was interesting in connection with the rather extensive history, the involvement of the cord and the facial muscles, and the effect of the radium as it had up to the present time been demonstrated. The use of the radium had been entirely without any unpleasant consequences, and there had been no reaction at all. The applications had been from about twenty to thirty minute periods. Dr. Harris said that he remembered a case which had been reported by Dr. Wilson, of Bridgeport, a number of years ago, where radium was used with equally beneficial results.

DISCUSSION.

Dr. Quinlan said he would like to ask whether any part of the growth adhered to the canal wall, especially the anterior and inferior part.

Dr. Harris said that when the case was first seen by Dr. Haskin, they thought they were dealing with an abnormal condition of the jugular bulb. The Wassermann reaction was negative, and the picture of the chest also.

Dr. McKernon said he would like to ask how many treatments of radium this patient had had, and at what intervals.

Dr. Harris said the treatments had been given at weekly intervals, eight last spring and four in the month of September.

Dr. McKernon said he would like to know if the mass in the canal had increased in size or changed in color.

Dr. Harris said there had been a shrinkage of the condition since radium was used from last May. The improvement had been observed by a half dozen men who had been following the case from that time.

Dr. McKernon said he would like to know the pathologic report. He had seen two cases, one external and one in the canal, very similar to Dr. Harris' case. In this case radium was used, and while there was a distinct cessation for a period of several weeks in the one case, and a period of several months in the other, they both had recurrences and died.

Dr. Phillips said some of the gentlemen present might recall the case of epithelioma which he showed before the society. In that patient the primary growth was in the floor of the canal, and a general practitioner had considered it a mastoid case and had operated on it. Later he had been obliged to reopen and clear out the entire area, which had then become involved. Dr. Abbey used radium on this case, and she suffered excruciating pain following its application. It never produced any effect on her, and the speaker did not know how many applications she had received, but thought three, four or five.

Dr. Berens said he would like to know if that mastoid was open when the patient received the applications of radium.

Dr. Phillips said this case was rather unusual. If the case presented was one of eipthelioma, it would be the youngest he had seen. The woman he showed was about thirty-nine or forty years of age, and had been the youngest he had seen. Clinically this case did not look like epithelioma but more like sarcoma.

Dr. Haskin said if he was not mistaken, the pathologist at St. Luke's Hospital stated at the time of the report he was unable to say positively whether the piece of the growth removed by the forceps was malignant or not.

Dr. Toeplitz said one interesting feature in this case was the paralysis of the left recurrent laryngeal nerve. The only explanation seemed to be that there was a metastatic growth which should be followed out, otherwise he could not see how the recurrent laryngeal nerve could be affected, since this nerve only went up as far as the omohyoid muscle. He remembered a case which he had seen with Dr. MacDonald, in which it had been difficult to make a diagnosis; the paralyses of the facial and of the laryngeal recurrent could only be explained by a carcinoma of the esophagus and a metastatic growth pressing on the facial, after it had passed the foramen stylomastoideum, from which the patient died three months later.

Dr. Berens said that while the patient was under observation he had asked Dr. Harris whether a Wassermann test had been made, and was told that there were three made, all of which were negative. The paralysis of the vocal cord, it seemed to the speaker, could be explained on the lines of syphilis in connection with this growth. There might be a syphilitic neuritis. Cases of syphilitic neuritis of the recurrent laryngeal nerve are not uncommon, and clear up very quickly under proper treatment. He had seen at least three, probably four or five, cases of syphilitic paralysis clear up under mercurial treatment before salvarsan had come into use, and two cases cleared up rapidly from the use of salvarsan.

Dr. Duel said that although all three Wassermann reactions had been negative, if syphilis was responsible for the growth, a positive indication might be obtained by spinal puncture and examination of the fluid. Many of these cases would not give a positive Wassermann reaction by blood test. The speaker had seen a case only that week in which there was undoubtedly syphilitic involvement of the eighth nerve, where examination of the blood failed to show a positive Wassermann reaction.

DR. BERENS said he was very glad that Dr. Duel had brought out the point about spinal puncture in regard to the negative Wassermann reaction.

DR. QUINLAN said the growth looked malignant in nature. Dr. Schwartz had told him a number of years ago that probably fifty per cent of the growths springing from the canal, and especially from the anterior and inferior canal wall, resulting from chronic suppuration, had more or less of a malignant aspect. The speaker, in his judgment, would be inclined, even without a microscopic examination, to term it malignant. The paralysis of the left vocal cord he did not quite understand about, as there was no evidence of infiltration or inflammation about that cord.

DR. HARRIS said there was absolutely no infiltration or inflammation about the cord.

DR. QUINLAN said if this was a malignant growth, was embedded in the tissue, and macroscopically had the appearance of a malignant growth, the main line of argument would tend towards the specific element of the disease. This growth in the canal had the appearance of a mixed growth, involving also the cutaneous structure and mucous membrane. He did not touch it, therefore could not tell how much resiliency was present.

DR. HARRIS said that the growth was very vascular, and that he had no further examination made since the one originally made at St. Luke's.

DR. QUINLAN said he had seen two such growths. One protruded externally in a man seventy-one years of age, who had had the growth for over fifty years, and had been advised by several surgeons not to have anything done. It was probably one of very serious nature, and ordinary manipulation by examination caused profuse hemorrhage. A small piece had been removed, and on examination it was found to be a benign growth. A postauricular incision was made, which showed it to be adherent to the posterior and inferior canal. This operation took place three years ago. Dr. Quinlan saw the man about four or five months ago, and there was no evidence that the condition had returned. The other case he had seen in Halle with Dr. Schwartz, who emphasized particularly the importance of a malignant diagnosis in growths springing from the canal wall. Dr. Quinlan said he would like to ask Dr. Harris to report subsequently on the case, and also the condition in the larynx.

Dr. Harris said the patient was originally treated for his ear, and the paralysis then occurred, one and one-half years ago. The voice was now perfect by usual compensation of the other cord, and the man was able to preach.

Dr. Eagleton said he had brought with him some X-ray plates to show metastatic carcinoma of the temporal bone. He wanted to ask Dr. Harris if he had any X-ray plates taken since the original X-ray. Since the first report of his own case findings, about two years ago, there had been so much progress in X-ray interpretation that he thought if Dr. Harris would have an X-ray taken now, it might throw great light on the nature of the growth itself.

Dr. Berens said he would like to suggest to Dr. Harris, if this patient objected to lumbar puncture, that no possible harm could result from the use of salvarsan. It was simply a point in the therapeutics and might substantiate the diagnosis.

Mastoiditis With Eczema and Atresia.

Dr. Harris said this case was simply illustrative of what is occasionally met with in these cases, and he wanted the gentlemen to see the picture presented. The patient, a young girl, had been under his care in the hospital for her nose and throat. There was a general eczema and an empyema of the antrum, frontals and ethmoids, which condition was now virtually well. She showed a suppurative process in her right ear, upon which a mastoid had been performed. In the left ear there was almost complete obstruction. The atresia did not permit the entrance of the finest probe he could use. A simple mastoid had been performed and later a radical operation. The gentleman who had performed it thought that the atresia had followed the simple mastoid. This case suggested the question regarding the propriety of doing anything to better the atresia. She had absolute loss of hearing in the left ear, and both vestibular and cochlear labyrinth were dead. She was still having a rather free discharge from the left ear and also from the right. The question was more than an academic one, because where there was secretion she had eczema.

Dr. Phillips said he would like to ask if there was any history of a period during which this patient had an active labyrinthitis.

Dr. Harris said there was no history of any data of a period during which this patient had active labyrinthitis. There was a dead labyrinth on the left side, but the trouble which required radical treatment was in the right ear. He said he could not get a probe through, but there must be a minute passage there, for the ear discharged freely on the right side.

Dr. Berens said the point to be particularly talked about was whether they should operate on the ear which was suppurating but could hear. The other ear was dead. The question to be decided was whether a radical operation should be done on the first ear.

Dr. Harris said he thought that this case was deserving of a very free discussion.

Dr. Quinlan said that the condition of this canal confirmed him more than ever in the thought that they should never allow anyone else to follow out their subsequent line of treatment after a mastoid operation. He remembered two cases where he had allowed the family physician to follow up the treatment by dressing the wound. Sometimes in these operations the assistants, in their zeal and enthusiasm, would drag the ear anteriorly. If the ear is not kept well back and upward, and support given to the upper posterior wall, there would be this relaxed condition. If they allowed the family physician to continue on the cases, they would get this stenotic condition, as has happened in the case first presented. Although there was no suppuration in his cases, one a boy and the other a girl, still there was a band of cutaneous tissue inside the scaphoid fossa between the drum and external orifice.

Dr. Dench said that an excellent meatus could be formed by a perfectly simple plastic operation. He stated that he would turn the ear forward and make the ordinary Dench flap. Undoubtedly some other form of flap would do equally well. He did not believe that there would be much danger of infection of the flap if the ear were kept thoroughly cleansed prior to the operation. It was to be remembered in these cases that local infection was not apt to occur, as the tissues of the patient were already pretty well fortified against such an infection. He would hesitate to do a radical operation on the ear which had the best hearing, but would suggest the plastic operation as a primary procedure. If the suppuration stopped, well and good. If not, he believed that a careful radical operation would effect a cure without much menace to the hearing. He would certainly do a radical operation upon the ear in which there was no hearing.

Dr. Macpherson said he would like to know if any of the gentlemen present had tried the new Dakin solution on any of these cases, which had been used so much in France for wound infections. He used it in two cases. One, an intermitting discharging ear, cleared up, but it had done so previously by another treatment. In the second, a granulation about the size of a pea, springing from the region of the attic, dried up after destroying it with one application of silver, followed for about ten days by the use of the solution.

Dr. Lutz said that he had used this Dakin solution to which Dr. Macpherson referred, in the middle of the summer, and found that in discharging ears it did very well. He dropped it in just as one did peroxid.

Presentation of Specimens of Labyrinth and Fetus.

Dr. McKernon showed two specimens for their beautiful mounting, which had been placed in a fluid. One showed a labyrinth very distinctly, and the only part which he wished to know about was the nature of the fluid, which was a secret one. He had obtained the specimens in Leipzig, and had been told that the temporal bone and labyrinthine structure was simply put into this fluid. The other specimen was a fetus. They had received no previous treatment whatever. He saw a large number of such specimens in the laboratory at Leipzig and had picked out these two. The tissue was taken off and they were then placed in jars.

DISCUSSION.

Dr. Dench said that he had seen several specimens from this same laboratory. He wished to know if the bone had not been decalcified first. In his opinion, the bone had first been decalcified and then dropped into the preserving fluid.

Dr. Berens said that Dr. Duel and he had done some similar work in 1904 or 1905, and the bone then was first decalcified, although these specimens were said not to be so treated.

Dr. Lutz asked if at the laboratory they had given Dr. McKernon the length of time it took to bring the specimens to that stage.

Dr. McKernon said they had not given him the length of time it took to bring the specimens to this stage at the laboratory. They must have had about fifty on the shelf, and these were put in the solution just the same as the two specimens he presented.

Carcinoma of Temporal Bone and Mastoid, and Presentation of Other X-Ray Plates.

Dr. Eagleton: The first plate taken was of an old gentleman, about seventy years of age. Fifteen years previously he had had a cancer of the lip, which was removed in the regular way, and then epithelioma of the side of the nose, which was removed by snow. He later had an epithelioma of the ear, which was excised. The one of the lobe of the ear and one on the lip were pronounced as basal cell epithelioma. Eight months after the last removal, he noticed he was becoming deaf and had a buzzing in his ear. Two months later he developed dizziness and nystagmus of the left side. Six weeks later there were a few drops of blood and pus from the ear without pain. The local physician diagnosed the case as one of abscess, but as the patient had no recurrence of the discharge and no pain, he refused the diagnosis and went to an aurist, who inflated him.

When first seen by Dr. Eagleton, about two months previously, he had a small granulating mass occluding the posterior wall. He was totally deaf, the vestibule did not react to the caloric test, and there was slight facial paralysis. Pressure over the mastoid, which was not tender, made him dizzy. An X-ray of the two mastoids was made stereoscopically, and the speaker said they ought to be seen that way to be properly appreciated. The diagnosis by the radiographer was carcinoma of the temporal bone, involving the whole mastoid region. Nothing could be seen by Dr. Eagleton except the small mass springing from the posterior wall. He made a large incision and found the skin perfectly normal. He then exposed it and found both the inner and outer plate filled to the dura. The wound healed by primary union, but three days previously the facial paralysis had become complete. The case was remarkable because it showed a metastatic bone carcinoma and not a primary carcinoma, which was so frequently seen. He had the primary growth there, and it was a metastasis not by direct extension. In looking over the literature on the subject, in the textbooks of otology, the speaker said they always mentioned the form of carcinoma which grew by extension and not metastasis. The rarity of the condition had caused him to look up the literature, and he was rather thoroughly surprised to find that only two-tenths of one per cent of skin carcinomas ever caused metastases of bone. Primary carcinoma of the thyroid, adrenals and kidneys caused a large number of bone metastases in different parts of the body. Those of the thyroid and prostate very frequently gave metastases of the skull and temporal bone.

The diagnosis in these X-ray plates was so interesting that the speaker went through some of his old plates. In regard to syphilis, he had some time ago had a woman under his observation who presented herself with what was thought to be a frontal sinus involvement. The X-ray plate taken demonstrated that she had a cranial syphilis just above the eye, although, according to her physician, he would not put it in its proper place. The erosion of bone looked very much like an osteomyelitis. It could be pretty correctly diagnosed as of a granulomatous nature and a diagnosis of syphilis was made Up to the time of the radiograph there had been no suspicion of it. She was practically going to be operated on in the frontal sinus by a New York physician when the X-ray was taken and a dose of salvarsan cured her.

Another plate showed a case of slow growing carcinoma of the antrum, and the blood was examined. A careful diagnosis was made by the radiographer, who stated the patient had periosteal spicules growing in irregular form in the growth itself. The speaker said it should be stereoscopically seen to be appreciated.

Another plate presented a rather rapidly growing sarcoma of the sphenoid. The difference between two X-ray plates could be seen. In the one there was no bone formation whatever. The man died, and the sphenoid and cranial cavity were found involved.

Another plate showed a new bony formation and tumor formation, which resulted from continued suppuration, and was a case of hemorrhagic periostitis. It was opened up and a section removed, from which a pathologic diagnosis was then made.

Dr. Eagleton said the case he wished discussed was that of the carcinoma. He suggested that if Dr. Harris had an X-ray taken now of the case he showed at the present time, no mistake would be made, and it would probably be found to be a rapidly growing sarcoma or carcinoma—likely a carcinoma.

DISCUSSION.

Dr. Haskin said he wanted to speak of the spicules in the floor of the antrum which Dr. Eagleton mentioned. The speaker was rather interested in some specimens shown by Dr. Cryer, who would take up a skull and state that it was a syphilitic skull from the peculiar formation of the inner surface, especially its anterior middle fossa. There were stalactites sticking up all through the surface of the bones. He showed Dr. Haskin about fifty specimens of his own, which he said were undoubtedly syphilitic. It was rather interesting, because the speaker, in his own recollection, had had three or four specimens in the sphenoid sinus, where from ten to fifteen stalactites were sticking up from the floor of the sphenoid sinus. He did not know what the history of these patients had been, or whether they would show up in an X-ray picture, but they were very marked. Dr. Mosher stated he had seen one or two spicules in such cases, but never such a collection in any one sinus.

Dr. Harris said he was sure Mr. King would have another X-ray picture taken and he would report the case at a later time.

Stenosis of Ears by Shock.

Dr. Quinlan said he wished to report a case he had seen at St. Vincent's Hospital, about three weeks previously, directly after the Hughes celebration in this city. The patient, a young man, was enthusiastic about getting a picture of Mr. Hughes, and had set his camera for the same. By some improper provision the spark, used in connection with his camera, blew up and the boy was thrown to the ground. He lay there unconscious for several minutes, when he was picked up and brought to Bellevue Hospital.

Dr. Quinlan saw the boy the next day, and found that both eyes were closed and it was impossible for the patient to open them, although by force some argyrol was put between the lids. The external auditory canals of both ears were absolutely stenosed. The skin was swollen, and it was only by using force that the drums could be seen, and then only partially. Both drums were torn, the boy could not hear, and, as he could not see, did not know what had befallen him. He went on in that condition for some time. The speaker saw him again a few days subsequently. All his symptoms were cleared up and the drum membranes were now plainly visible. There was no congestion or flush of inflammation about them.

The shock had been so intense as to do so much damage, and yet repair had taken place within a very short time. The boy now heard a whisper at a distance of four feet. The ears were in fairly good condition, except that he had more or less tinnitus, which was troublesome, especially in the left ear. The drum in this ear was still open in one place and the rent showed up, and there was an area with a slight inflammation around the aperture which was subsiding. It was rather curious to have found the eyes and ears in such an awful condition following shock.

DISCUSSION.

Dr. Berens said he would like to ask Dr. Quinlan whether the upper tone limit in this case was lowered.

Dr. Quinlan said that the upper tone limit in this case had been lowered, but that it had come back.

Dr. Harris said he would like to ask Dr. Quinlan whether he had recognized any labyrinthine condition in this case. He made the suggestion, because a large number of such cases were presenting themselves abroad, as the result of shell explosion in the great war. These cases were the result either of direct concussion or of injury. The former were apt to get well. The latter of course remained permanently deaf.

Dr. Quinlan said that he had at first thought the shock might have caused extravasation of blood in the labyrinth, but on account of the rapidity of the clearing up process he was inclined to think it resulted only in mere concussion.

Dr. Dench said he thought the concussion due to explosion was the cause of the impairment of hearing and lowering of the upper tone limit. He asked if there had been any facial paralysis. Continuing the discussion, Dr. Dench said that he understood that the external auditory meatus was found closed, and would like to ask by what. He asked if the camera blew up and set fire to the film.

Dr. Quinlan said that external trauma closed the ears. The condition looked like an extensive erythema. On the whole, the features could not be distinguished at all. There was an extensive burn affecting the entire cuticle. In trying to get the picture he had been thrown from his position by the explosion. Dr. Quinlan said he did not know what caused the accident. The patient said it was probable that some sparks dropped in the pan attached to the camera, and his only regret was that he could not replace his camera, as the one destroyed came from the other side. There had been no facial paralysis, and the patient was all right now, except for a slight perforation in the left drum membrane.

Two Cases of Mastoiditis With Staphylococcus Organism.

Dr. Phillips said he wished to report two cases which he had seen this fall. The first was in a man, sixty-five years of age, who had come from Montana, who had a large postauricular abscess and all the evidences of advanced mastoiditis. In the latter part of May, in some way, a spiked grass, known as foxtail, had gotten into his canal and injured the ear drum. A day or two afterwards there was a discharge. A paracentesis was done. There was continuous discharge from then up to the time Dr. Phillips saw him. He complained of mastoid pain, but the suffering had not been severe. The local otologist who cared for him did not consider the case one of mastoiditis, but at the time the man left he had no postauricular swelling. The speaker operated on him, and at that time, of course, he had tenderness as a result of the periostitis. The entire mastoid process was found involved and full of granulation tissue and pus throughout the entire area. The tegmen was absorbed and quite a large area from the knee of the sinus. The interesting point in this case was that examination of the secretion from the ear showed it to be a staphylococcus aureus.

Dr. Phillips said he would not have been so much impressed with the case but for the fact that within three weeks from that time he saw a girl, from a near-by boarding school, who had been sent to him for an abscess of the ear. There was a discharge with very little pain or pus, but a continuous temperature of ninety-nine and one-half degrees every day. A paracentesis was performed. Some slides had been made from the condition, and a report of staphylococcus aureus was returned. The young woman never seemed quite ill, but at the same time the disease in the mastoid process progressed steadily. There never was very much tenderness, but the speaker never saw a time when she did not have a slight temperature, and the area of involvement seemed to increase. A radiograph was finally made, for she had been suffering about three weeks, which showed a very extensive mastoid involvement. She was operated on and granulation tissue was found throughout the entire mastoid area.

Dr. Phillips said in a very large experience in mastoid cases he did not recall many with this peculiar infection. He was tremendously impressed by the extent of the disease, and had come to think that these were cases one had better not postpone too long. The girl made a remarkably rapid recovery and never had any reaction from the operation, and about the ninth day he permitted her to leave the hospital and take a train for her home in Detroit. He wondered whether any of the gentlemen present had had much experience in cases of this type.

DISCUSSION.

Dr. Rae said he would like to ask Dr. Phillips whether the bacillus in these cases had been obtained by smear or culture.

Dr. Phillips said that a culture of both these cases had been made by Dr. Dwyer.

Dr. Dench said that he considered staphylococcus infection very rare. He had seen a number of cases in which the predominating organism in the smear was staphylococcus, but in operating upon the mastoid the mastoid smear showed streptococcus. He believed thoroughly in Dr. Phillips' suggestion as to prompt operation upon cases of suspected mastoiditis. The opening of the mastoid was such a simple procedure, and the cases made such perfect recovery after early operation, that in a case where there was a doubt the mastoid should be opened early.

In speaking of Dr. Quinlan's case, this case illustrated how quickly recovery followed exploratory operation upon the mastoid. He cited a case coming under his own observation in which there had been a rupture of the drum membrane following a fall. Suppuration had ceased when Dr. Dench last saw the case, but in spite of this fact the mastoid was opened. A small area of suppuration was found, but a fracture of the skull was discovered at this time, the line of fracture passing vertically downward through the squamous plate and entering the mastoid. The line of fracture was explored and an extradural clot evacuated. The entire wound was sutured, excepting at the lower angle, where a small drain was inserted. The man was well at the end of seven days, the wound being completely healed. The patient had epileptiform convulsions prior to operation. His epilepsy had disappeared after the operation.

Dr. Duel said that the study of the staphylococcus albus was very much in its infancy. At present the custom in the laboratory was to classify as staphylococcus albus all cultures of cocci which did not produce pigment or hemolysis. The virulence of these organisms was very variable, and he thought the time would come when the various strains would be differentiated in some such way as the pneumonia organism is at present. Some of them were perhaps most virulent, while others were benign. Some studies of cases of staphylococcus albus infection had recently been made by Dr. Dwyer at the Manhattan Eye, Ear and Throat Hospital Laboratory, which would excite much interest and might lead to important differentiation of these organisms of great clinical value.

Dr. Quinlan said he would like to ask Dr. Dench if it were his custom, when he had a fracture of the skull, with profuse discharge from the ear, to irrigate. Was there not danger of carrying the infection into the cranial cavity by so doing and filling up the fissure with infective material?

Dr. Dench reiterated his belief that there was not the least possible danger of infection being carried inward by irrigation. He did, however, believe that there was a positive danger if the external auditory canal was allowed to remain full of discharge. This was particularly true in traumatic cases where there had been a fracture of the skull with rupture of the membrana tympani.

Dr. Quinlan said he would ask Dr. Dench if he used suction in these cases and then wiped dry. He said force might carry infection through the fracture into the labyrinth. It was purely a thought regarding these fractures accompanied by suppuration, as to what was the wisest provision, in these cases, whether to wipe by absorbent cotton or by irrigation.

Dr. Dench said that some method must be employed by which the majority of these patients could take care of themselves. They could not use a suction apparatus, and he believed that irrigation of the external auditory canal was the most feasible method of keeping the canal clean. He could conceive of absolutely no danger following this plan of treatment. He said that as the intracranial pressure was positive, irrigation of the ear could not possibly force microorganisms into the subdural space. He realized that at the present day the treatment of acute suppuration by irrigation was questioned. He called the attention of the Fellows, however, to the fact that this plan of treatment had withstood the test of time, and he believed it to be the most feasible means of keeping the external auditory canal sterile.

Dr. McKernon said, in regard to the cases Dr. Phillips spoke of, that some years ago he had a case, four and one-half years of age, in which an incision was made into the drum membrane, a smear taken, and the report of staphylococcus albus made. Some four or five days later the child developed a mastoid, and after that the speaker took the culture down to Dr. Sondern. He also reported staphylococcus albus in the pus. Some eight days later the child developed sinus thrombosis. Dr. McKernon resected the vein, and the clot in the vein for about one inch below the foramen was cultured and showed a pure streptococcus. He agreed with Dr. Duel's idea, that they would know more about this later on. Oftentimes an infection in the mastoid on culture proved more virulent than when the culture was taken from the lesion. He did not believe that staphylococcic infections, as they saw them, were at all uncommon from the standpoint of mastoid infection. Those who were present at that meeting might recall the case reported by Dr. Day, of staphylococcus infection in the mastoid, where the mastoid was involved to such an extent that it was almost beyond treatment. Dr. McKernon said he had seen several cases of pure staphylococcus, both in the smear and culture, but those cases in which the jugular was involved usually showed in the culture of the clot in the vein a distinctly pure streptococcus.

Dr. Dench said that early this year he had seen a child who apparently had had a very mild middle ear infection. About five days after the inception of the attack, and when Dr. Dench first saw the case, the patient had a temperature of 105°. The left ear showed signs of a mild acute otitis media. The left drum membrane was incised, and a smear showed cocci in groups and occasionally in pairs. A blood culture was made when the drum membrane was incised, and twenty-four hours later showed streptococcus in pure culture. The mastoid was opened, the lateral sinus opened, and the jugular excised. The patient made an uninterrupted recovery.

Dr. Dench called attention to the fact that the exact report as to the character of a smear depended very largely upon the particular laboratory to which the smear was sent. In one of the hospitals with which he was connected, the smear from the external auditory canal almost always showed staphylococcus, and it was only upon culture that the true infecting organism was determined. In another institution, where perhaps more time had been given to the study of aural infections, the laboratory report of the smear taken from the canal almost invariably showed the organism which when the case came to mastoid operation was found to be present in the mastoid secretion. For this reason Dr. Dench believed that in reports of aural smears the personal equation of the bacteriologist could not be neglected.

In closing, Dr. Dench said that in his own experience he had seen only one case of pure staphylococcus infection of the mastoid.

Dr. Duel said he thought that an important advance was soon to be made in the differentiation of the different strains of the staphylococcus albus.

Dr. Phillips said that these cases made him feel a great deal more afraid of that type of infection than he had been before.

Report of Case Previously Presented.

Dr. Lutz said that the case which he had reported at the last meeting of a girl with apparent meningitic symptoms and a sterile fluid turned out to be a case of poliomyelitis. He had two such cases at the Brooklyn Eye and Ear Hospital.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of March 20, 1917.

THE PRESIDENT, DR. STANTON A. FRIEDBERG, IN THE CHAIR.

Case of Falsetto Voice.

DR. ELMER L. KENYON presented a young man who had possessed a falsetto voice, but whose voice was now normal Dr. Kenyon said that this patient had been first presented two or three months previously, when his voice was of the falsetto type. The falsetto voice had developed when he was fourteen years old, without any special determining circumstances. There had been no essential trouble in getting back his natural voice, and with an intelligent patient there is usually no great trouble. The greatest difficulty had been that the patient had at first been unwilling to accept the new voice. He could not believe it was the kind of voice he should have. Dr. Kenyon said the present voice was not as perfect as it should be, because the patient had not been able to give sufficient time for training; when one remembers that the falsetto voice results from a psychologic misconception that develops into a fixed habit of holding the larynx high up in the throat under tension, one realizes that it may take time to overcome the habit fully. If one remembers these causal factors and knows something concerning the mechanics of the voice, he should quite readily be able to bring about correction. The speaker's habit was to explain to the patient the main facts concerning voice production, to demonstrate the unnatural high position of the larynx, point out the unnaturally high pitch and tenseness of the voice, and so lead up to the training which causes the voice to rest on chest instead of on throat action. The voice showed great improvement since the last time he was shown. Dr. Kenyon said he had hoped to bring a patient who not only had a very high pitched voice similar to this, but also a markedly tense voice, in whom the problem was much harder.

Dr. Kenyon also presented a young man who stammered.

The patient had hesitated about coming over, because he said it would be very hard for him to talk before an audience. The patient began to stammer when he was a small child, his father having also been a stammerer. He was twenty-six years of age, and had previously made six efforts to recover, and had been treated in four other large cities in the United States in an attempt to get relief. He had taken treatment at two of the best known and largest schools for stammerers, and had spent more than four months with a neurologist in New York City. He came to Chicago on December 14th and went home on the first of February under complete control He had returned for the greater security of a more fully developed technic. Dr. Kenyon said he was not "cured" (no stammerer was ever cured in three months), but he had control over his speech, which, if kept up long enough, would ultimately result in cure.

Dr. Kenyon said that stammering schools which advertised so extensively, practically all "guaranteed" a cure in six weeks, but that in fact a "cure," in the very nature of the disorder, was utterly impossible in so brief a time, unless it happened that the stammerer was just on the point of spontaneous recovery. However, it was often possible for the stammerer to gain a capability of control in a less period of time. Some of these advertisers who promise such lavishly impossible things have the written published endorsement of leading physicians. In other words, these men who get their living by deliberate deception are, in some instances, backed up by reputable physicians, who themselves are doubtless deceived. Such competition was hard to meet by ethical methods. The patient talked under perfect control, explaining that his four months of treatment under psychoanalysis had resulted in absolutely no benefit. He had stammered worse at the end than at the beginning of that treatment.

Carcinoma of the Larynx.

Dr. Joseph Beck presented a case of carcinoma of the larynx, and showed the specimen and microscopic section.

Dr. Beck said one of the interesting points about it was that the patient had been under treatment by X-rays for two years and was apparently cured by that method of treatment. The specimen showed that it was quite a large growth and required a laryngectomy. He said it was unusual to find such a case in the anterior part of the larynx within the boundaries so it might be removed entirely before the invasion had extended to the neighboring regions, and for this reason he considered it interesting. He said the point he wished to make about the after-care was in reference to the voice as a question of an artificial larynx, and he prepared this patient for such a proposition. He had left an opening in the subhyoid region which he thought would ultimately be lined with mucous membrane and would allow the upper part of the artificial larynx to be introduced behind the tongue and the other end into the trachea. He had introduced a rubber tube in that way, and the patient could speak so that he could be understood across the length of a room, and at the same time he could breathe through it freely. He said that was the great feature about laryngectomy; the patients fear that they will be dumb, and although they are told that they will be able to make a sound with their buccal cavity, it is not that of speech. He intended to consult Dr. Kenyon about producing the artificial voice as had an artificial larynx inserted into the trachea had found sidered it too early at present, for it had only been but two months since the operation. Dr. Beck said that most cases who soon as the patient had reached the proper stage, but he con- them unsatisfactory and had thrown them away. He said someone had devised an artificial larynx in which one end fitted into the trachea and along the side of the lower jaw and side of face into the nose and back into the nasopharynx. In that way the patient got a voice. That apparatus was supposed to be the best that there was known at present. He said the tube in this case presented was not objectionable, because it was hidden. By bringing the chin against the chest the hyoid bone would compress the tube and thus prevent the food from going down into it, but rather allowed to pass into the esophagus. The possibility of his having a voice was most interesting.

Radium in Sarcoma of the Larynx.

Dr. Beck also presented a case showing the results of the use of large doses of radium in sarcoma of the larynx.

He said the case offered many points of interest in the history, but he would only state that he had had two tracheotomies in order to save his life. He had a rapidly growing tumor for which he had a thyrotomy with the burning process of Percy's method, cleaning out the whole anterior portion of the larynx. There resulted a recurrence and a great sloughing from the burn. They had followed this up with large doses of radium. Dr. Beck thought the profession was very fortunate in having in Chicago an institution where they could have the use of such large doses of radium. He had always been doubtful about the value of radium, because in such amounts as he could use it had not proved beneficial, at least not in carcinoma. The rapidity with which the sarcoma melted away in this case was simply marvelous.

Sarcoma of the Hard Palate.

Dr. Beck showed some stereoscopic photographs of the case. The points of interest were the rapid disappearance of a tumor the size of a Lima bean and the occurrence of a severe burn of the tongue and palate. The amounts used were 125 mgs. for twenty-four hours in eight hour periods. In the laryngeal sarcoma the patient had 75 mgs. for thirty-six hours at twelve hour intervals.

DISCUSSION.

Dr. Elmer L. Kenyon thought Dr. Beck's scheme for producing the voice was very ingenious and likely to bring about excellent results. He said he would be very glad indeed to watch it. He thought it was true that patients with carcinoma who had had a laryngectomy and had used a laryngeal tube of the old type soon discontinued its use, but that a certain amount of them, with patience and perseverance, could develop a voice of low carrying power, and some of them occasionally were able to develop a comparatively large voice. He saw no reason why Dr. Beck's scheme was not a very good one indeed.

Hemorrhagic Mastoiditis.*

By George W. Boot, M. D.

DISCUSSION.

Dr. Joseph C. Beck said the case of his that Dr. Boot reported gave none of the classical symptoms or indications for a mastoid operation except the fact of the brain symptoms, which made him decide to operate even as late as he did. He thought that Dr. H. W. Loeb had reported two cases instead of one, and referred to this other one; his bacteriologic examination disclosed the diplococcus. He also called attention to a possible connection between that and pneumococcus. Dr. Beck did not know that a bacteriologic examination had been made and a definite result obtained in his case, but remembered they had made cultures at the time of the opening of the mastoid. He thought the most interesting thing was the X-ray pictures, and said that the mastoid cells in this case showed them to be clear, and he thought that the light over the mastoid probably would not transilluminate the mastoid process. That made the X-rays of more value than the transillumination, as they were not obstructed by the blood clots. He said he had worked a little further on this case—namely, on the pathology of the mastoid bone. He said he had sectioned this bone and was going to give Dr. Boot the result of his histologic examination. Grunnert was one of the first to bring out the subject of phlebitic osteitis of the mastoid cells, and called attention to the fact that in those cases sinus thrombosis was common, either by way of the emissary vein or through the inner plate directly to the sinus. This case of his was not of that type. This was not an osteophlebitis septica of the mastoid, because the bone was not affected. The pathology was a great deal like the mastoid in which the cells are filled up with material which pulsates and bleeds very easily. He thought the work that had been done on the confluent mastoiditis would be remembered, where all the partitions were destroyed by the pressure of that material. Dr. Beck thought it was very interesting to study the pathology of mastoid disease after operation, the same as the study of X-ray pictures after operation; he stated that he liked to study the X-ray pictures after operation because one often saw things then that they did not see before, and could use it in subsequent cases.

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*See page 744.

Dr. J. Holinger cited his observation of a lady, sixty-four years of age, who was very delicate and became sick suddenly one Saturday afternoon. The house physician made what he called a paracentesis, but the patient grew worse. She had some fever, which was to be expected in a person of that age. Dr. Holinger saw her on Monday morning. She had suffered severely all day Sunday and Sunday night. Had received a hypodermic of a quarter of a grain of morphin at four o'clock in the morning and an eighth at seven o'clock. When he saw her at eight o'clock she was in severe pain. There was some discharge from the ear and a few blood clots which he could not remove. The morphin had practically no effect, although she had received three-eighths of a grain, which was certainly a considerable dose, especially, as became evident later on, as she was very susceptible to narcotics. She had vomited and there was definite spontaneous nystagmus. Dr. Holinger made a diagnosis of beginning invasion of the labyrinth. He said two members of the society saw her, and promptly denied the presence of meningitis. According to postmortem statistics, the diagnosis of meningitis is made by far too seldom. Therefore, in spite of the absence of a Kernig, he insisted upon an operation on account of the symptoms of irritation of the labyrinth. At the operation, two days later, the cells which were distant from the antrum were clear, the antrum and middle ear were full of tightly adherent blood clots similar to those Dr. Boot described. The patient felt greatly relieved after the operation and made a good recovery.

Dr. Holinger asked whether there were any other patients of this kind showing symptoms of irritation of the labyrinth in connection with hemorrhagic mastoiditis.

Dr. A. S. Rochester said that within the last few hours he had operated on a case which, while he did not believe it was a pure hemorrhagic mastoiditis, still it presented some features which made it of interest in connection with this paper. The patient had had a cold in the head four days previously, used a nasal douche, and within a few hours developed an acute pain in both ears. The drum membranes were found to be bulging, and a paracentesis was done on both sides; on the left there was a very bloody serous discharge, and on the right side a slightly bloody discharge, the blood soon clearing up. The left continued to be bloody constantly, but the right showed very little blood. He had today become convinced that both mastoids should be opened, and so had opened them both this afternoon. The left mastoid, which had the bloody discharge, during the operation bled profusely, the cells were broken down and filled with very bloody granulations and pus. On the right side there was very little bleeding during the operation, the cells were broken down and filled with yellowish pus but very little blood. He thought it was interesting, because from the start the one side was so bloody and the other was not.

Dr. Charles H. Long thought a number of such cases would be found if they were looked for more carefully. He said that in working in the laboratory he had found two or three cases, but knew nothing about the history.

Dr. Joseph C. Beck, in answer to Dr. Long, said that Wingrave had shown postmortem changes in the sinus as well as in the mastoids, the same as Dr. Long had found.

Dr. Boot, in closing, disagreed with Dr. Beck about what Dr. Loeb reported; he said Loeb distinctly called the organism a diplostreptococcus. He thought there was a close relation between these cases and pneumonia, and the fact that the exudate in the cells was a fibrinous affair, not serous, and the fact that there was very little bleeding was interesting. He thought it was a distinct type of mastoid, and not very common.

Hemorrhage in Rhinolaryngologic Work.*

By Arthur M. Corwin, M. D.

DISCUSSION.

Dr. J. Holinger thought there should be a differentiation between hemophilia and simple bleeders. He thought that hemophilia was a well characterized disease, and appeared to be well known to the people themselves, and that it existed here. He stated that he knew a family with two sons who both died from hemorrhage—one from a simple little pinch of the finger between the doors. The finger bled and bled and could not be stopped. He thought people were often careless about telling the physician that there was a hemophilic tendency in the family, and in one such case a physician removed the tonsils. The hemorrhage was not very strong in the beginning, but twenty minutes afterwards there was a distinct gush, and within five minutes the boy was dead. He said Dr. Corwin had stated that there were few grown male hemophiliacs, and drew from that the conclusion that the abnormality was overcome later on; but that was a mistake, for the reason was that they died young from slight injuries. He said the doctor who lost a tonsil case was Dr. Banga, and he had worked on the very problem in Europe and was the first to bring out the peculiar feature of heredity, that daughters of hemophiliac fathers would have hemophiliac sons, while they themselves were not hemophiliac.

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*See page 749.

The speaker was sorry that Dr. Corwin did not follow up the literature on coagulin, and stated that in Prof. Kocher's clinic in Berne an assistant, by name of Fonio, had for years followed up these cases of slow coagulation, especially the cases of hemophilia, and found the cases of hemophilia had a lessened number of blood platelets. The platelets were not entirely absent, but were much more rare than in the normal blood. Fonio gave as an explanation of hemophilia a congenital scarcity of blood platelets. Dr. Holinger said he had shown the coagulin (Fonio) in this society. It consisted of the blood platelets isolated from the blood and treated in such a way that they did not deteriorate, and their effect on a bleeding surface was identically the same as the effect of the blood platelets in the blood. The blood platelets deteriorate very readily as soon as the blood is exposed to the air. This sets free a ferment which causes the fibrin to coagulate. Fonio succeeded in stopping hemorrhage in hemophiliacs by means of coagulin.

Dr. George W. Boot said that in cases of hemorrhage with a high blood pressure, he thought it was a good plan to give the patient a hypodermic injection of nitroglycerin to lower the blood pressure. He also believed that hemophilia occurred in this country. In one case at Cook County Hospital a year or so ago, where a tonsillectomy was done, they tried for two days to stop the bleeding, trying horse serum, transfusion of blood from one of the parents, and everything else. He said the test of the blood for coagulation was very simple, they only needed a wire with several little loops in it. The successive loops containing blood could be dipped into water at half minute intervals until at the end of five minutes it was possible to tell whether the blood would coagulate promptly or not.

DR. CHARLES M. ROBERTSON thought that socalled hemophilia in most instances was transitory. He believed the so-called hemophiliac was one in which the condition of the blood was the result of some infection, and changed with conditions of the patient at different times; a patient that was hemophilic today might not be hemophilic a week or a month from now. He thought the cases which were bleeders from birth might inherit this habit, but that even those might be cases which were infecetd in utero, in which case they may overcome the condition. He said that Dr. Lespinasse had operated on fifty cases under one year of age, and some as low as four days, by means of a transfusion by the direct methods, and this had stopped all of these bleeding cases and saved the children. He said he had noticed in many cases in operating upon the nose that there were certain ones in which the action of adrenalin was peculiar; the patient would be treated with applications of adrenalin with absolutely no effect at one time, and at a later period the effect would be good. That, to his mind, was an indication that the patient in the first instance was in the throes of an infection, and when his condition was improved the coagulability of the blood assumed a normal condition. He thought they should be careful in stating that there were many hemophiliacs; there was no doubt that there were a few who were true hemophiliacs, but even in these cases they must eliminate the condition of the blood which might be produced by a chronic infection, and we know that nearly all cases which come to us have infection, in the alveolar process, the tonsillar region or in the gastrointestinal tract, so in dealing with hemophilia it had been his custom to treat it as a symptom rather than a real condition.

DR. HARRY KAHN thought that the time of coagulation, seven, three, or ten minutes, depended upon the method, and that each method must be standardized. He said there were many methods, but the simplest was to take the watch, puncture the lobe of the ear, put a drop on the watch crystal and then with a rounded needle, wait until you could draw out a thread of fibrin, time the procedure on the watch; this usually occurred in three minutes. He advised the use of a round needle rather than a cutting needle, so as not to cut up the blood platelets or corpuscles. He thought no matter what method was used, it should be standardized and the same methods used in all cases; probably none of them showed the real coagulation of the blood, but in long coagulation time, eight to ten minutes, he had had no bad effects from operation. The hemorrhage had stopped just as promptly as in shorter periods. Cases in which longer time was required for the coagulation should not be operated, but be treated and held until the coagulation time was reduced to normal limits.

Dr. Harold I. Lillie said that the research laboratories of Leland Stanford had produced very interesting work on the blood. They had shown that calcium lactate, if administered preoperatively, in a good many cases is ineffective except in one condition, and that was chronic jaundice, in which the calcium was firmly bound when it did increase the coagulability of the blood. They further say that the five, ten or fifteen minutes as a normal time for the coagulation of the blood to take place could not be named definitely, because, as Kahn suggests, the methods differ and have not been standardized, and unless the coagulation could not take place up to an hour, it might be said to be within normal limits. He said they had also shown by animal and patient experimentation that the sera on the market if more than a month old was ineffective. It must be fresh. Human serum is very much more efficient. The whole blood can be administered subcutaneously or intravenously much quicker than to wait for the serum. In a report from Budapest, where thirty-five cases of spontaneous hemorrhage in children occurred without any definite clinical sign, cultures from the nose showed pure cultures of diphtheritic bacilli. These cases cleared up promptly with massive doses of antitoxin. The speaker said he had one such case this year where the hemoglobin was forty-five per cent, the patient had been bleeding at the nose for two days, and appeared extremely ill, and when the pack was removed he continued to bleed. There was no clinical evidence of any diphtheria—a little thin discharge, which no one could suspect as diphtheria. Culture from the nose showed diphtheria bacilli. The patient was given 30,000 units of antitoxin; in three days' time a double otitis media developed. Incision of the ear produced a very thin pus which was also shown to contain the typical diphtheria bacillus. This cleared up within four days.

He said this case occurred in private practice, and the only symptom was nose bleed.

Dr. Joseph C. Beck thought the discussion would be profitable, particularly from the point of nomenclature, for the loose way of applying the term hemophilia. Some of the men stated tonight that they had seen cases of hemophilia in this country. He had been carefully watching for a clear case, from the point of the history, of hemophilia, but thus far never encountered one. He believed one important point when they were to operate on a case was the history of that particular condition. He was sure the question of bleeding was frequently overlooked, and the history of bleeding should be gone into more carefully. He thought men did not often ask if there was profuse bleeding in the family following extraction of the teeth or other minor things.

He said Dr. Corwin had stated that adrenalin was often ineffectual on account of raising the blood pressure rather than decreasing it, but there were types of hemorrhage in which adrenalin was a specific, especially in cases of hemorrhage of the eyes, nasal and skin bleeding of the newborn. He had seen two cases in the newborn in which nose bleeding and mouth bleeding was present, where the use of fifteen minims of adrenalin was repeated every two hours and the bleeding stopped, and the child had remained well now for three years Dr. Beck was sure the adrenalin must have an effect, probably from its compensatory action, and he thought this point should be made in exception to the statement that it was ineffectual.

The speaker thought Dr. Corwin had brought out very forcibly the fact that the term "hemophilia" is used loosely. He thought it was often applied in place of cholemia, and said that excessive bile in the blood would retard the coagulation greatly, and the same was true in septic conditions.

The speaker thought that in cases where the question of diphtheria arose, that when the diphtheria bacillus was found and the antitoxin used, one had a right to believe that it was the antitoxin that stopped the bleeding, but he believed the serum in the antitoxin was also of value in stopping the bleeding. He said one of the first cases on which he used serums for bleeding was a case which had been bleeding for five days after a nasal operation; the temperature was 106° F., the patient had received salt solution under the skin, and a double infection resulted, one due to the bacillus aerogenous had developed. The hemoglobin was very greatly reduced and the blood pressure was low. In this case he had stopped the hemorrhage by a large dose of antidiphtheritic serum. He believed it was a question whether in these cases the bleeding was not stopped by the serum rather than the antitoxin. He also cited the case of a lady who had a postpartum hemorrhage following the third child birth, the first two children having produced no trouble. In the time between the birth of the second child and the third one she had an operation for necrosing pancreatitis, and following this she had several attacks of rectal bleeding which were diagnosed as internal piles. Following this third delivery she had, as stated before, a postpartum hemorrhage, and was a long time recovering from the effect of the bleeding. Three months later he was called to see her on account of a nose bleed which had been very profuse. There was no evidence of a bleeding point in the nose, but on the body, around the knees and some other places, there was evidence of small hemorrhages (Shoenlein's disease). Dr. Herrick saw the case in consultation, and concurred in that diagnosis. Dr. Beck said this patient lived for a year with hemorrhages which were best controlled with placental blood locally applied; if fresh blood is locally applied it will often cause a hasty coagulation. Professor Hiss, who was in this country at that time and saw the patient in consultation, advised her going to Europe to be treated, and the family sent her there. There she received injections of large doses of radium solution, and following the injection of these radium solutions, which were gigantic, the bleeding stopped almost instantly, no matter from where it occurred. Nevertheless, she died from this prolonged bleeding, which was diagnosed as purpura hemorrhagica.

The speaker said that two of the children during the last year developed attacks of acute otitis media. They were suffering from tonsil and adenoid most profoundly. The family doctor insisted that as soon as they recovered from the ear trouble the tonsils and adenoids be removed, but Dr. Beck refused because of the history of the mother. But later on they took up the question again, because he felt it was only fair to the children that those tonsils and adenoids should be removed. They found that one child gave a coagulation time of seventeen minutes and one thirteen minutes, consequently they put them on calcium salts, and inside of a month they determined, by the Sahlie method, the clotting time to be seven and six minutes respectively. They decided then to operate under nitrous oxid and ether anesthesia with a very little accessory ether. The tonsils and adenoids were removed without much bleeding, but he sewed in gauze sponges in each tonsil cavity and there occurred no bleeding. When the tampons were removed there was also no bleeding, and none after that. Dr. Beck did not know whether the calcium had the effect or the tamponage, or whether the children would not have bled anyway, but it was an interesting fact. The internists believe that the removal of the tonsils from these children will have a tendency to protect them against the development of such a condition as occurred in the mother.

Dr. Corwin, in closing, said that Dr. Holinger evidently must have misunderstood him in referring to the fact that there were few adult hemophiliacs. He knew that it was true that there were few of them, but it was perfectly possible for a true hemophiliac to outgrow the proposition if they were kept alive long enough. He thought very little was understood about this condition; the blood picture was largely normal, but the blood clotting is markedly below normal, and the blood platelets, according to many authorities, are not reduced as they are in purpuric conditions. Why bleed? Faulty clotting. Why faulty clotting? Nobody knows. So far as the platelets are concerned, the speaker thought it had not been proven that their lack counted at all. He considered the coagulum good as far as horse serum was good, but not so good as coagulose which was made of human blood.

He said Dr. Boot brought out some good points, but while nitroglycerin might be used to lower the blood pressure, pituitrin would do the same thing, as he had noted in the case of three adults who had extremely severe bleeding and where pituitrin had lowered the blood pressure, though commonly supposed to raise it.

He thought that when Dr. Robertson said that hemophilia was sometimes hereditary, he was wide of the mark, for it was always hereditary—more so than any other disease known; the doctor had also said it was transitory, and if he meant by that that it usually kills people quickly, he was right about it, but if they survived by the use of horse serum or what not, they might recover and live to adult years.

He thought the points brought out by Dr. Lillie and Dr. Beck were excellent ones, and he believed the history of heredity and the personal history of the patient should always be considered, as a single violent bleeding is not to be taken as a hemophilia, under any circumstances, without other considerations, and one of those was the history of female transmission only to males. He said there were no female hemophiliacs. In the case of the lady with the pancreatitis with a developed hemophilia, he thought that brought out the very thing he wished to make definite in the paper, and that was that there is a great grouping of purpuras—many varieties included in a symptom complex—without any such definite pathologic background. The superficial hemorrhagic spots and the deep seated, the simple purpura and the purpura hemorrhagica, are only different in degree, and all mean toxic conditions with a leakage through the vessels because the vessels themselves are deteriorated, and also blood lysis, as is not the case in true hemophilia. In hemophilia there is neither blood deterioration which we can prove nor vessel deterioration, but in the children and in the case of the lady there was a blood lysis and vessel deterioration.

Dr. Corwin said the point of the paper was to bring out the sharp distinction as between a proposition which was always hereditary and one with practically normal blood picture but greatly delayed coagulation, and all the other purpuric conglomerate forms of hemorrhage which are present today and absent tomorrow, and which have no place in the classification of hemophilia.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of April 17, 1917.

THE PRESIDENT, DR. STANTON A. FRIEDBERG, IN THE CHAIR.

Perforation of the Septum.

DR. JOSEPH C. BECK presented a patient who had a perforation of the septum as large as the end of the thumb, the etiologic factor of which was not proven. There was no evidence of its being syphilitic, but it had troubled her greatly because of ulceration of the edges. Dr. Ballenger had attempted to heal it by transplantation of cartilage, but without success. Dr. Beck had subsequently made several attempts, one by transplanting the middle turbinate body into the space, but without results. He had afterward succeeded very well in closing the perforation, bringing forward the ends of the inferior turbinate body, the posterior end on one and the anterior end on the other. At the time of exhibition the left anterior body passed through the anterior part of the perforation into the right nostril and sutured to the edge of the margin of the perforation. All that remained to be done now was to cut the pedicle half off the posterior end of this turbinate before placing it with the margins of the perforation. The patient was presented to demonstrate that large perforations may be closed. This was the first one of this size that Dr. Beck had succeeded in closing. He had also used the inferior turbinate body in a similar way in closing a cleft in the hard palate which would have been very difficult to close otherwise. He found that the inferior turbinate body would heal in beautifully if its edges were made raw. He expected to finish the operation on this patient and then show the end results. The transplant acted as an obstructing agent at present, but this would not be the case later when it was trimmed down to the desired size and form. The question has often been asked of Dr. Beck, why go to all that trouble in order to close a large perforation that was not causing any more trouble than a slight crusting? He wished to state that the main reason was because the patient objected to the question "Have you ever had syphilis?" every time she had a nasal examination by a different man.

Subperiosteal Abscess of the Mastoid: Cure by Paracentesis.*

SAMUEL SALINGER, M. D.

DISCUSSION.

DR. OTTO J. STEIN said it had always seemed remarkable to him that we did not see more such cases as Dr. Salinger described. Probably a good many are seen without being recognized. He had observed a similar case a good many years ago which led him to think that the abscess originated through the squamous mastoid, which is recognized as the most common avenue through which pus can reach the surface of the cortex aside from perforating through the bone. In this case he discovered that the abscess had developed by dissection along the roof of the auditory canal. He saw another case a few years later, and therefore this route should be taken into consideration as an avenue along which pus may reach the surface of the cortex by dissection along the canal wall, its entrance being through a defect in the tympanic ring or via the Rivinian notch, and in that way produce all the manifestations of a perforation through the cortex. In such cases where the drum has not ruptured it is very feasible that resolution may result without operation on the mastoid.

DR. JOSEPH C. BECK thought that a thesis usually presented all the facts a man could gather, especially on such an important matter as this, and the fact that the essayist's case got well would be good evidence that that was good treatment. Mention was made in the paper of statistics by men like Hakaza Kanasugi, who has done many more operations than was mentioned. Kanasugi is an expert anatomist on the mastoid, he has supplemented his studies with X-rays of the skull and has found many of these of great interest. The essayist in his paper said nothing in reference to making a diagnosis by X-ray. Another statement was made, namely, that there are no mastoid cells until a certain time in life. This is constantly mentioned in older papers in the literature, but it is not borne out by operations on infants, many of whom have large mastoid cells. The roentgen diagnosis of the mastoid proper in cases like this which the doctor reports is very important. Just because these two cases recovered without operation at this time is a question whether it is as well as if an incision had been made and the antrum opened up. It is an unnatural drain of the deep parts of the opened mastoid and cannot drain well through the antrum and into the middle ear. In such cases as the doctor described if it is only a passage of the pus through the sutures the X-ray would show it.

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*See page 758.

Dr. Beck had also noticed the absence of names of two great workers along this line, one Luck and the other Kerrison. He asked for Dr. Salinger's opinion of these two papers.

Dr. Salinger, closing, said the point made by Dr. Stein was very well taken. The pus may reach the cortex by means of the auditory canal, but according to Mygind there are symptoms along the canal which would point to that; however, in his own cases those symptoms were not present.

Regarding the pneumatization of the mastoid, Dr. Beck misunderstood him. He said that according to the authors who studied it, pneumatization was not complete until the age of puberty, but that did not mean that there were not mastoid cells before that age. As to the X-rays, he was very unfortunate; one case was treated at home and the other was shipped out of the hospital before he had an opportunity to do much with it; the X-ray would undoubtedly have shown the existence of the sutures.

Report of a Case of Nasal Sarcoma Cured by Radium.*

OTTO J. STEIN, M. D.

DISCUSSION.

Dr. Albert Woelfel said that however one might evaluate this report as evidence of a case of sarcoma cured by radium, it was at least a very good example of what is known by radium users, namely, the peculiar susceptibility to the action of radium of sarcomas of the upper respiratory passage. It is thought that round celled sarcoma generally responds to radium treatment more readily than other forms, but it is also an observation of radium users that tumors that are nominally of the same type show a different response to radium treatment in different parts of the system. This case is more than a good example of the ready response to radium of this kind of a tumor in the upper respiratory passage, however, it is one of the best to illustrate this point that we know of. When he recalled the difficulties in the application of radium and other circumstances in this case, he was much surprised when Dr. Stein reported the end result.

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*See page 782.

Another thing the case illustrated was the efficiency of distance irradiations. Physicans often say they know that if you can apply radium directly to malignancies you can effect something, but for distance irradiation it is no use. But in this case we certainly have an illustration of what distance irradiation can do. In this patient where there was bulging and fixation in the abducted position of the eyeball, due to invasion of the socket, there is no doubt that the mobility of the eye which followed the irradiation around the orbit in three or four weeks was effected by the radium.

Another point the paper and his experience with Dr. Stein on this case illustrates is the mistaken idea that seems to prevail that large doses of radium are necessary to effect such changes in sarcomata. Under the influence of the reports of Kelly, who describes irradiations with enormous doses of radium, a physician said to Dr. Woelfel recently: "I don't believe you fellows know anything about radium," because we prescribe such relatively small doses. We have been puzzled by the reports of Kelly regarding the use of such large amounts of radium, and the only way we can account for it is that he must be using the emanation from these large amounts of radium, which emanation would have quite a different radiating power, unless it is drawn off when the radium is in equilibrium. If such large amounts of radium were applied for any time at close range they would have a burning effect that would hardly be beneficial.

The final point was that in using radium we should not be so impatient of results. Dr. Stein told how, before we thought we were through with the patient, further radium treatment had been prescribed. It was not believed that the irradiation that had been employed had had so much effect. It takes time for the effect to develop, a fact that is being learned more and more. Probably therefore the best general technic to adopt is to begin a series of irradiations, get the gross effect and then wait for some weeks to finally get after the treatment to effect the end result.

DR. JOSEPH C. BECK said he had two cases of sarcoma which had been treated by Dr. Woelfel by those large doses of radium. One was sarcoma of the hard palate, the other of the larynx. The tumor of the hard palate was the size of a bean, flattened on the surface of the hard palate. He excised a piece for microscopic examination and found same to be a sarcoma. This patient received one hundred and twenty-five milligrams for twenty-four hours in three different treatments, and following that went home. He returned in less than two weeks, when the tumor had entirely disappeared, but he presented a very severe burn of the tongue and of the soft palate, from the action of the radium. This burn was very painful (presented pictures showing burned area). He thought this accident should be mentioned. The doctor used a screen of a rubber material which contained some metal, which was a mistake and would not happen again. The patient is apparently cured of the sarcoma, two months having elapsed since the treatment.

The other case was the sarcoma of the larynx which was presented at the last meeting; the sarcoma simply melted away. There were very few sarcoma cells in a recent section and very little remains of the tumor. He was to have more radium treatment and he hoped he would be cured of the sarcoma, which had been removed twice by external and internal operations and had also been burned by the Percy cautery, which he wished he had never used in any case.

Another case, a congenital lymphangioma in a little girl, had grown considerably recently. Microscopic section had verified the diagnosis of lymphangioma. The little girl had seventy-five milligrams of radium on April 16th and one hundred milligrams on the 17th, and he hoped they would have the desired effect. He showed photographs of the case.

As to large and small doses of radium he showed a photograph of another case of angioma on the upper lid and which spread in six weeks to a great extent. He had used CO_2 snow in the forehead, injected the main mass on the cheek with boiling water and used ten milligrams of radium pure over the eyelid. The snow removed the angioma but left an ugly scar and so did the hot water injection treatment, but the end results from radium were very satisfactory.

Another case he showed was a papilloma which involved the entire interior of the larynx and which he removed under suspension. The patient now has a mass of papillary excrescences around the tracheal opening. He intended to use radium in this case, for it has been shown that good results are obtained in the cases.

It must not be forgotten that sarcoma is greatly influenced by the X-ray also. Deep penetration treatment by X-ray will have almost as good an effect as the radium in many cases. Arsenic should not be neglected as a method of treatment of sarcomas, used in the form of salvarsan or cacodylate of soda.

DR. ROBERT SONNENSCHEIN said that some years ago he had an opportunity while working in Koenigsberg of reporting the longest standing case of sarcoma of the nose on record. The patient lived for eleven years after the diagnosis was made. It has been shown that sarcoma of the nose is particularly benign as compared with tumors elsewhere. He did not know whether that had a bearing on the cases mentioned here in regard to the efficiency of the radium and X-rays, but it may be that the character of the tumor in this location is such that it is more amenable to treatment than elsewhere.

DR. CHARLES H. LONG said the radium treatment in these cases was the one to discuss, but in a case he wished to mention the important thing was the diagnosis of the tumor. About the year 1910 a small boy came to Mercy Hospital for removal of adenoids. These were removed and the boy left for home, but returned with hemorrhages in the throat. Dr. Zeit examined the specimen and pronounced it sarcoma. The patient was brought to Dr. Long and they had a special tube made to use X-rays. He thought there were only three treatments, and at each treatment a severe hemorrhage took place and severe pain in the head, so the mother refused a continuation of the treatment. The parents expected that the child would die, but he kept in touch with the case until 1911, when he was hurriedly called to the house, as the boy was supposedly dying of suffocation, but the patient had all the symptoms Dr. Stein spoke of—the bulging of the eye and cheek, the lowering of the hard and soft palate, etc., a long narrow piece of dark colored flesh lying parallel with the tongue was observed which seemed to have an attachment in the throat. This was pinched to see if it was dead, but found that it was very sensitive and movable, only twisted on its pedicle and had become strangulated. He sent the child to the hospital and had another examination made, and the report was fibroma, while the previous report had been round celled sarcoma. In looking up the literature he had found it was difficult to make a positive diagnosis between these two conditions in their early stages of development. In 1911 they removed the tumor, which was larger than a goose egg and had many tentacles besides the one in the mouth. It could not be removed through the nose, so they removed it through the mouth by incising the soft and hard palate, encircling the pedicle of the growth with a wire snare, then resecting the roots which were widely attached. The child made a good recovery.

Dr. C. W. Hanford was interested in the case, as he had seen the patient and given a few applications of radium in the early days of the treatment. He thought well to start with one hundred milligrams and quickly diminish it to about forty milligrams, and he believed it could be decreased even more than that. He preferred not to see carcinomas of the tonsils and base of the tongue, for he thought no amount of radium, small or large, would do any good, except to add possibly a few months to their life and make their existence somewhat more agreeable. In cauliflower growths on the soft palate it sometimes requires great ingenuity to have something to hold the radium in contact, so he sent the patient to the dentist to have an appliance made. It happened in two cases that the upper teeth were out of the way and a plate was made to fit the gum, and in the back portion he vulcanized a strip of wire in the rubber, which could be twisted around the tube so it could be put directly against the growths. By this procedure these growths melt away very rapidly.

Dr. Stein, in closing, said he had been interested in radium for some time in the socalled inoperable cases, but had always received a great deal of discouragement from those who had used it, and he had used it in this case with a great deal of misgiving—first, because of the discouraging reports, and, second, because it was almost impossible up to this time to get a sufficient amount of radium. He had decided that radium to be of any value would have to be used in large quantities properly applied. He realized the very well known fact that sarcomatous growths in the nose sometimes disappear spontaneously, but nobody had ever offered any explanation for it. There is possibly still room for doubt as to the value of radium, but since the growth in this case had disappeared under the application of radium, and they were fortunate enough to be able to prove its disappearance by the postmortem findings, he felt justified in thinking that the radium had produced the change.

He had been rather surprised to hear Dr. Beck speak so favorably of radium, as he had discouraged him greatly in the past. When he asked Dr. Beck about using radium in this case he had said he thought it would be of no value; that while the patient might be benefited there would be a recurrence and not a cure. Perhaps the doctor's change of opinion had been brought about by the use of larger amounts of radium—he had always said that the radium he had at hand was probably not of sufficient amount, and it might not have been applied properly either. He would hesitate to take the improvement of any of the cases Dr. Beck reported this evening very seriously, as they were evidently very recent cases. He thought a longer period of time should elapse before such cases were reported, and something more than merely improvement in the condition. The improvement is interesting, but we want something definite—whether there is any real value and by what means.

The question as to the amount of radium to use is a very important one, and it is only recently that we have had access to such large amounts.

The point brought up by Dr. Hanford he considered very interesting. He had read some reports in a French medical journal on the treatment by radium of sarcoma of the nose and mouth by the means just spoken of, and he thought radium could be applied in that way in a more intelligent manner. Many surgeons working around the mouth have devised some really wonderful appliances by which the radium could be applied directly to the growths. There is much to learn about radium, and a case of this kind is highly instructive.

Dr. Joseph C. Beck, replying to Dr. Stein, said the reason he (Dr. Stein) was able to make such a definite report in his case was by reason of the untimely death; had that not occurred he could not have made such a complete report. He thought the criticism offered of his report was not well taken. The report was not made as final, but he thought in such cases it was well to make a report from time to time. The case of palatal tumor had not returned after a lapse of two months, but may still recur. He always made the report as the basis of his experiments. Ten years ago he reported on his negative results with what he thought was radium, at least he bought it for radium, but it was not pure. He had subsequently used ten milligrams of pure radium, which was as much as he could afford to have in his possession at that time. Neither of these amounts was sufficient in such desperate cases as cancer. Since we had been fortunate enough to have access to these large amounts, he had gone to work again and was reporting this present result. Under no circumstances did he wish, by his reports, to discourage any one from experimenting, but he believed in being very critical in the use of these new methods of treatment, since truth will out.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of May 22, 1917.

THE PRESIDENT, DR. STANTON A. FRIEDBERG, IN THE CHAIR.

Papilloma of the Larynx.

DR. H. L. POLLOCK presented a little boy aged six years who had been referred to Dr. Joseph C. Beck with a diagnosis of a papilloma of the larynx. When he was one year of age he became hoarse and gradually became worse until he was three. He was then taken to a physician, who did a laryngotomy and removed a growth from the larynx, the nature of which was not known. Two weeks later he was taken back on account of difficulty in breathing and a second laryngotomy was performed. At this time there was so much difficulty in breathing that a tracheotomy was also done and a tube inserted. The boy had worn the tube until he was brought to them a few weeks previously, and it had been in his throat for a year without removal. (Exhibited photographs of patient with tube in position.) They (Drs. Beck and Pollock) removed part of the external papilloma and curetted the entire larynx. Two weeks later the balance of the papilloma was removed.

The tracheotomy tube was left out and the patient observed; as the breathing was perfect, the tube was not replaced.

The doctors intended to do a plastic operation to close this tracheotomy wound. The patient's condition was very good and he was able to use his voice slightly.

(Note: A plastic operation has since been done, closing the external tracheal opening, which healed promptly. The child was kept under observation for several weeks, and as breathing was perfect and voice returning he was allowed to return home.)

DISCUSSION.

DR. A. H. ANDREWS said it was quite usual for papilloma in these young children to return. He thought it would be exceedingly fortunate if the tumor did not recur before the child was grown.

Dr. Joseph C. Beck thought there was a great difference in the removal of a papilloma in former days by the indirect method and the method of suspension by which this work was done. This was not a simple papilloma; the histologic examination showed a great deal of granulation tissue.

Dr. Pollock, closing, said that over two years ago they had a similar case from the same city in which the same operation was performed. They removed the growth by means of the suspension method and the voice was much better. It had been two years since the papilloma had been removed and there had been no recurrence in that case.

Foreign Bodies in the Esophagus: An Analysis of Fifty Cases.*

Stanton A. Friedberg, M. D

DISCUSSION.

Dr. Robert Clyde Lynch, New Orleans, stated that his experience in extracting foreign bodies from the esophagus had been similar to that of Dr. Friedberg in all instances. About three weeks ago he had had the unique experience of seeing into the mediastinum through the esophagus of a child three and one-half years old, who had swallowed lye, which had produced a perforation of the esophagus. Through the perforation he was able to recover from the mediastinum a pint of fluid, water, etc. The child lived for about six hours and died from the infection of the mediastinum.

He believed that in about five per cent of the cases he had seen with foreign bodies in the esophagus the smooth ones would go through; rough, sharp edged bodies stop behind the cricoid, while meat balls land at the cardiac end of the esophagus. He cited the case of a physician who would not wear false teeth who was called into the country to see a patient and was given the privilege of eating some Louisiana chicken (which did not grow nice and tender as they do up here). The doctor ate a leg and second joint of the chicken, and not being able to grind the shreds into small bits, a ball of undigested chicken was arrested at the cardiac end of the esophagus and it took an hour or more to pick out those threads. Crab shells very often lodge in the esophagus, and they have the peculiarity of producing no obstruction to the swallowing of either fluids or solids. In five cases none had had any difficulty, and in each instance he had recovered the shell, but in many cases it was necessary to split the shell and break it into fragments before it could be removed so as not to traumatize the parts. In some of the cases the suspension apparatus had served them very well. By creating a breath-way with a rubber tube (not a catheter, but an open end rubber tube), passed into the trachea between the vocal cords and then practically suspending the esophagus and lifting it up, it will give an opportunity to work further down in the esophagus with both hands and in that way use two pairs of forceps, which facilitates the removal of impacted foreign bodies. Occasionally there will be little fragments that will have to be picked out. One patient swallowed a pin and the head became buried in the cartilage at the base of the epiglottis with the point sticking into the esophagus through the mucous membrane over the arytenoid cartilage so that it was brought tight in such a manner that he could not swallow or move his neck, and did not have water or food for a period of about thirty-six hours. With an esophagoscope they looked down and the first time went by the foreign body, but then he suspended him and there was the pin beautifully displayed, pinning the posterior portion of the larynx to the esophagus very nicely and it was removed without any trouble.

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*See page 768.

The speaker thought the symptoms of the presence of foreign bodies in the esophagus were very misleading to a man who had no experience with these cases, for many of them give no obstruction to swallowing, simply producing an increase of secretion of the mucous membrane of the upper portion of the esophagus and a slight irritation from this which overflows from the esophagus into the larynx and worries the patients because they have to cough to get rid of it. Most of the patients have the feeling that something is wrong without any definite symptoms at all. He believed that where there was any history of swallowing a foreign body, that was sufficient to demand an examination of the esophagus for its presence or absence.

DR. NORVAL H. PIERCE said he had a great sense of comfort in knowing that Dr. Friedberg was living in Chicago, where he could be called upon at any time for his special skill. He had seen him work and knew his work was as good as anyone's in the country.

The last case Dr. Pierce had been called upon to examine was one at St. Luke's Hospital, a Belgian refugee, who was employed as a governess in Chicago and came with the history of having got a foreign body in the throat during a meal composed largely of Illinois chicken. She complained of great pain. She was rather a neurotic type and had undergone a great deal of suffering in her native country. There was nothing to indicate anything in the esophagus except the pain on swallowing; no excess salivary secretion, etc. He saw her in the office late in the afternoon, sent her to St. Luke's and saw her there in the evening after X-rays had been taken. The pain was in the region back of the cricoid cartilage, but nothing showed in the radiograph. There was a shadow, but the X-ray man was not positive whether it was a foreign body or the cartilage of the larynx. He went to the hospital the next morning and found that the patient had left, having coughed out a triangular shaped piece of bone the base of which would perhaps measure an inch and a half. He cited this case simply to accentuate the fact that we cannot depend upon any symptom as pathognomonic of a foreign body in the upper part of the esophagus, nor can we depend always upon the X-ray. He believed that when a patient had swallowed a foreign body the best plan was to search for it, but he had seen a great many cases where nothing had been done and the symptoms had all disappeared within the course of a few days. He thought the safest course was to search for them rather than to allow them to go on until inflammation occurred and caused increased difficulty in removing the foreign body.

DR. FRIEDBERG, closing, emphasized the necessity of always making a careful examination, stating that it is extremely difficult to determine whether anything is present or not. In the soft tissues swelling occurs rapidly and hides the foreign body, so it is likely to be overlooked. As to the time that should be spent in looking for a foreign body, he considered anything over half an hour in a child or young person harmful, believing that it was better to make a short examination and repeat it later if necessary, as they could not stand the shock well.

Suspension Laryngoscopy.*

H. L. POLLOCK, M. D.

DISCUSSION.

DR. ROBERT CLYDE LYNCH, New Orleans, said regarding suspension under local anesthesia that he had maintained that suspension was preferable under general anesthesia until Dr. Israel had suggested powdered cocain, which was now used in the great majority of adult cases. He thought this had added greatly to the comfort of the patient under local anesthesia, and there was so far no systemic effect of the cocain used in that manner. In his practice he had practically done away with the necessity for an elaborate table; any ordinary table top which is strong enough to hold the crane would suffice. He no longer drops the head over the edge of the table as he first described; this has two advantages—first, that it is very much easier for assistants who are not accustomed to seeing suspension done or to help in a suspension, and very much easier for the physician or anyone to hold the head straight when it is resting on the table. Secondly, instead of putting the crane at the very end of the table it is put back as far as the horizontal portion of the crane will permit, with the handle of the crane simply swinging over the edge of the table; try to get the patient up in the Johnson position for a view of the larynx rather than in the extreme extension that the old position demanded. The old position demanded that the operator look up along the line of the spatula, and the new position permits looking down along the spatula. This has lessened the tension that occurs on the neck and hyoid bone. Under the old position this region becomes white and in the new this does not occur, and there is also less tension on the upper teeth. The only disadvantage he could find with the new position was that the lower teeth must be watched a little more carefully to see that the spatula did not press on them too hard. To guard against injury to the lower teeth he had recently been covering them with a modeling compound.

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*See page 793.

Referring to the work on the epiglottis, the speaker said that in cases where he suspected lesions of the epiglottis, such as papillomas in children where the face of the epiglottis has some of the growth, he places the spatula on the base of the tongue and then raises the jack, forces the tip of the spatula down the lingual face of the epiglottis and then extends the patient, in that way obtaining a very good view of the whole face of the epiglottis and bringing into view the portion wherein the epiglottis lies above the anterior commissure of the vocal cords. That presents a very much more normal position of the vocal cords; they are not stretched so much as when the spatula takes up the place under the epiglottis, and in cases under cocain it affords a better opportunity for operating. In cases where he deliberately goes in for epiglottis work he uses a shorter spatula than for larynx work and drives it down along the base of the tongue, thus obtaining a better view.

He had had thirty-three cases of simple papilloma in children; not the pedunculated growth seen in the young adult, but the multiple papillomatous growths that have always recurred after removal. In the thirty-three there were thirty cases that they had operated at one sitting and there had been no recurrence. They had used no medicine or application that would tend to inhibit a new growth, depending upon the operation alone to determine whether the dissection was complete or not. In the three cases in which recurrence had occurred, the first was in one where he operated away from New Orleans, and he thought the fact that he was not at home accounted for the recurrence. The second time he resected in his own home, and the growth had remained away for a year. One of the other cases had been operated on six times and the other four times. The one operated six times has finally come to a tracheotomy. In that case the growth apparently extended down beneath the mucous membrane, and the child now has a larynx that is as hard as wood, fibrous, and yet there are true papillomatous growths down in the connective tissue. The microscopic sections could not be reported upon, as the last operation had taken place only three days previously. The other case was dissected perfectly clean macroscopically, so far as could be seen. The child did so beautifully under the anesthetic that they searched for papillomatous tissue but found no remnant at all. This child has regrown papillomatous material, not so rapidly as before, but if one was not careful the growth would come away with the forceps rather than give one an opportunity of dissecting the mass away from the base. The child was operated about six weeks previously, and there was no obstruction, although the voice had not returned to its normal. He considered the case in which the tissue was so thick and hard very interesting and thought it bore out the remarks of Green of Boston, who dilated on the fact that papilloma infected other tissues. In this case the child has grown papilloma of the glottis and of the uvula and at the junction of the supratonsillar fold which it never had before. The growths are small and pedunculated with a small pedicle and come away very easily. He believed the growths had taken hold along the line of the preceding operative procedures, and thought this bore out the fact that papilloma were in a manner contagious from one part of the tissue to another.

He had recently had a man who had laryngeal tuberculosis with quite a large ulcer involving half of the larynx and eating down into the arytenoid space so as to involve the cartilage, which would fold down and partially obstruct his breathing. After careful examination by an internist he declared that the process in the chest was thoroughly arrested. His epiglottis had been removed in Denver. They suspended the man under a general anesthetic and he had considerable difficulty with respiration; from the cyanosis that developed the flap became so edematous that it finally blocked his breath away entirely, so that they had to do a tracheotomy. They continued the anesthetic through the tracheotomy and then laid the ulcer open and found it was about three times the size they thought it was on the first examination. They cauterized this area with a most gratifying result, leaving the tracheotomy tube in place for a week until the area was healed, when they removed the tube and reestablished his breathway. If he had a similar case he would not wait to have to do a rapid tracheotomy, and thought it was perfectly justifiable to do a tracheotomy, for the anesthetic would allow one to do all they wished at one sitting without subjecting a tubercular patient to a second anesthetic. This man was suspended twice, and on the first period of suspension he developed symptoms of asthma with a rise of temperature and had rather a stormy convalescence following the first anesthetic. He had a period of rest for six weeks and then a second anesthetic, and he had the stormy time under the anesthetic, and the tracheotomy was done, but they succeeded in doing all the second time that they wanted to do the first time. He thought the man was perfectly well now, and the tracheotomy had done nothing except permit doing all that was necessary at the second sitting.

Dr. Joseph C. Beck stated that when he first employed suspension laryngoscopy he had the head flat on the table. At that time he had never seen the procedure performed, had seen no pictures or anything else, and simply placed the patient flat on the table. With the patient's head flat on the table he attempted to see upwards, but could see nothing. Since he had recently seen Dr. Lynch in Memphis do a suspension operation with the head on the table he had tried it again, but could not see as well as with the head hanging over the table edge, so he continues to do the operation in the latter way.

He considered the contagion or spread of a papilloma very interesting and said that the little boy exhibited by Dr. Pollock had a perfect papilloma around the tracheotomy wound which was attached to the skin.

Dr. Pollock, closing, said in reference to having the head flat on the table, that they had tried this position in a good many instances, but still could not succeed in getting as good a view as with the head hanging over the table. They had had no bad results from this position, as a surgical nurse always held the head. The longest time spent in operating had been forty-five minutes under extension, and they had had no bad results from that.

Dr. Pollock thought that if Dr. Lynch obtained such good results with the supine position there must be something wrong with their technic, and they would try his method again.

In regard to treating papillomas, they had used no medicine or cautery or anything of the kind in these cases, but in the carcinomatous or tuberculous conditions they had cauterized the area. In a great many of the cases the question of doing a tracheotomy arises and in many instances they do this previously. With the exception of very small children they are done under local anesthesia, thus obviating two anesthetics, but in children the general anesthetic is necessary. Most cases referred to them come with the tracheotomy tube in position. In one case of a little child on whom he attempted suspension there was a small opening from a previous tracheotomy wound and the child was becoming asphyxiated, so they opened this old tracheotomy wound and gave the anesthetic in that way. In one case in which there was large tonsils they did the laryngeal work first and then the tonsillectomy with practically no anesthetic. The child talked and made an uneventful recovery.

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LIV.

BONE METAPLASIA IN THE TONSIL.*

By

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HISTORICAL REVIEW.

The question of bone in the tonsil is of interest, not so much because of its clinical significance, but because the question of its possible origin has aroused widespread discussion, from the time Orth[1] reported the first two cases in 1893. Since then many reports have been issued by many different investigators, the opinion growing that the occurrence is not at all uncommon. In 1895, Deichert,[2] a pupil of Orth's, elaborated upon one of Orth's cases, described two of his own, and added in a footnote that two more had come to his attention since his article had gone to press. The year following, Sterling[3] found three cases, and in 1898 two Englishmen, Walsham[4] and Wingrave,[5] each reported two cases. In 1901 Pollak[6] renewed interest in this subject by publishing his findings of bone in many different types of glands, including tonsils. From that time until 1905 there was a long series of investigations, especially by German pathologists—Nösske, Ziegler,[7] and Töpfer[8] wrote in 1902, Reitman[9] in 1903, and the next year Carter,[10] Lubarsch,[11] Newcomb,[12] and Rückert,[13] followed by Halkin[14] and Schweitzer[15] in 1905. After that, interest was sporadic. Anselmi[16] reported in 1907, Lorenz and Grosvenor,[17] jointly, in 1908, and White[18] in 1911, Theodore[19] in 1912, Grünwald[20] in 1913, and Cary[21] in 1916.

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PATHOLOGY IN GENERAL.

Bone in the tonsil has been met with in the following forms:

1. There may be an extension of bone from the skeleton into the tonsil. In all such cases the surgeon has to cut through bone before the tonsil can be removed. Thus, Deichert reports in one of his cases, which also showed bone and cartilage nodules, the concommitant extension into the tonsil of the left styloid process, which had to be cut at enucleation. In the same case the hyoid bone was somewhat elongated and the right stylohyoid ligament contained a piece of bone two centimeters long. Rückert describes a case where cartilage in the tonsil was traced to its connection with the hyoid bone, Newcomb an extension of the styloid process into the tonsil, and Sterling three cases, in all of which the styloid process was probably responsible. The significance of these cases in the general controversy will be discussed later.

2. Another condition is the ossification of the capsule of the tonsil. Theodore describes a bony plate in the capsule of one of his cases; Lubarsch mentions a hardened capsule, and White reports one case in which the whole capsule is ossified.

3. By far the commonest condition is the finding of minute nodules of bone or cartilage or both in tonsils. They are usually microscopic in size, although in a few instances they are large enough to be seen with the naked eye. In almost every case they are confined to the connective tissue, only rarely encroaching on the lymphoid tissue. Walsham describes one case containing cartilage only, in which the nodules originating in the connective tissue penetrated the lymphoid tissue, almost to the surface epithelium. It is very common to find cartilage and bone in the same tonsil.

In order to determine, if possible, the origin of the bone and cartilage in the tonsils, it is necessary to determine the following points:

1. The relation of the bone and cartilage to the surrounding tissue and the presence or absence of a periosteum or perichondrium.

2. The relation of the cartilage and bone to each other, and whether the cartilage at any time appears to give rise to bone.

3. Whether the growing point of the nodule appears to be in the center or at the periphery. If in the center, it is less likely to have originated from the surrounding connective tissue.

4. Whether the surrounding tissue shows any evidence of inflammation or of other conditions that would be likely to lead to metaplastic changes.

Deichert, Nösske, Pollak, and Rückert have all reported cases where the cartilage or bone distinctly grades into the surrounding tissue. One of Deichert's cases shows the cartilage running into the connective tissue by thin threads. Nösske mentions a close connection between the connective tissue and the cartilage and bone, without giving details. In the same case he describes bone nodules originating partly from cartilage, but chiefly from the connective tissue. Pollak's case contained the bone in scar tissue, and was in intimate relation to it. In Rückert's, some of the nodules of cartilage were surrounded by a well-defined layer of connective tissue, while others graded into the surrounding tissue. Cary describes some of the bony nodules as being imperfectly separated from the surrounding connective tissue.

His cartilage showed a transition to a spindle type of cell, which blended with the connective tissue. Recently a case similar to this one came to our attention.

It was not always easy to determine when a periosteum was present. Rückert reported a case where the bone was retracted from the connective tissue, as if definitely separate from it, but there was no membrane surrounding it. Halkin mentions a case where fibrous connective tissue surrounded the bone in a manner resembling a periosteum, but he would not call it by that name. Theodore actually claimed to have seen a periosteum. In the case of cartilage nodules, Orth reports the finding of a perichondrium once, while Rückert claims to have seen it in every one of the seventeen cases of cartilage in the forty-eight tonsils of infants he examined. In one of his adult cases some of the nodules were surrounded by connective tissue in a manner similar to Halkin's bone case, while others graded into the connective tissue. Halkin says that in his case the cartilage seemed to act like a foreign body, spreading the connective tissue fibers apart, although no definite perichondrium was formed.

Osteoblasts were not reported with certainty in any case. Rückert thought he saw them in one case, while Halkin believed there were not only osteoblasts, but osteoclasts, with resorption of bone in one of his. Haversian canals, lamellæ, and bone corpuscles were much more common. Rückert suspected bone marrow in one of his cases, while Theodore simply describes open spaces filled with round cells and blood vessels. Cary simply says that some of his bony nodules "contained cells corresponding in morphology and position to osteoblasts and osteoclasts."

Most of the cartilage described was of the embryonic type, containing large cells in active division with but little ground substance and no fibers. Elastic fibers were described by Rückert in the cartilage of three cases, by Lubarsch in one fetus and in an infant.

The manner of ossification, where it could be determined, was a matter of interest. By far the largest number of investigators, when they mention their findings at all, describe ossification from cartilage. Among them are Rückert, Halkin, Theodore, Orth, and Deichert.

Rückert describes the gradation from large cartilage cells to small, bone-like cells. Theodore found every gradation, from pure cartilage to pure bone, in the same tonsil. His staining methods showed the zone of calcification. The youngest cartilage was in the middle, a fact which he interpreted to mean that the impetus for growth did not come from the surrounding connective tissue but from within the nodule. Halkin found in one of his cases evidence of ossification extending from the center, and from what he regarded as periosteum at the periphery. In the former case the young bone at the center was completely surrounded by the cartilage. Because of the presence of both types of ossification, Halkin concludes that the formation of bone is not a metaplastic process. He holds that under his conditions, where cartilage was transformed into bone, one would not be likely also to find a periosteum. Cary found both cartilage surrounding bone and bone surrounding cartilage.

The presence or absence of inflammation in the tonsil is an important point in the controversy, as will be seen later. Pollak, Lubarsch, Rückert, Walsham and Wingrave all worked on tuberculous patients. In some of these there was definite evidence of tuberculosis in the tonsils, whereas in others, although the tonsils showed inflammatory changes, there was no definite evidence of tuberculosis. One of Lubarsch's cases was an infant. Cary's case was from a woman who was a victim of recurrent tonsillitis. The enucleated tonsil gave evidence of inflammation by the presence of leucocytes and plasma cells.

THEORIES AS TO THE ORIGIN OF CARTILAGE AND BONE IN THE TONSIL.

Barring the cases where the hyoid bone and styloid process have been elongated in such a manner as to allow them to extend into the tonsils, two explanations have been advanced as to the origin of bone and cartilage in the tonsil. The first is that through pathologic changes in the tonsil the connective tissue has been so changed that an impetus to development in an abnormal direction is given. The bone and cartilage are, therefore, the result of a metaplastic process. The second is that during embryonic development, cartilage cell rests from the second branchial arch, close to which the tonsils originate, have been included in the "anlage" of the tonsil, and these, after a quiescent period, perhaps of many years, give rise under proper influences to the bone and cartilage nodules.

The embryonic theory was first set forth by Orth and Deichert, later by Walsham, Wingrave, Ribbert, Ziegler, Reitmann, Rückert, Lorenz and Grosvenor, Anselmi and Halkin.

Pollak, Nösske and Schweizer are the chief exponents of the metaplasia theory. All of them found degenerative changes, chiefly tuberculous, in the tonsils which they studied. The reasons they gave for their belief were the following:

1. The tonsil develops after all, or practically all, of the second branchial arch has been resorbed.

2. The cartilage is always found confined to the connective tissue, never in the lymphoid tissue. Even in Walsham's case, mentioned above, the nodules originated in the connective tissue, and merely penetrated the lymphoid tissue.

3. The nodules were usually found in such large numbers that it seemed improbable that there could have been so many rests of the arch.

4. Cartilage and bone were found only where there was distinct evidence of past or present inflammation.

5. There was a regular connection between the cartilage and bone and the adjacent tissue in their cases.

6. All cases reported up to the time of their studies were found among adults.

EMBRYOLOGY OF THE TONSIL.

In order to understand the first argument, it is necessary to know how the tonsil develops. It originates from the wall of the sinus tonsillaris, situated in the dorsal angle of the second pharyngeal pouch, the evagination of tissue between the second and third branchial arches. In the middle of the third month the sinus wall folds in two. From the outer coat and floor of each recess spring epithelial buds, at first solid, but later hollowing out and growing into the surrounding connective tissue, giving rise to the crypts. The tonsil has assumed its characteristic shape before lymphoid tissue is formed, the exact origin of which is doubtful. Hammar[22] maintains that it forms from connective tissue. Just previous to the formation of the lymphoid tissue, there is a marked increase in the amount of connective tissue. (See Figure 1.)

Within each arch lies a shaft of cartilage, those of the first three arches giving rise to various bony structures by ossification. From the second branchial arch arise the stapes, the styloid process of the temporal bone, and the lesser horn of the hyoid bone. Between the styloid and hyoid the cartilage degenerates to form the stylohyoid ligament. The length of the ligament is inversely in proportion to the length of the two bones which it connects, and which are variable in different individuals, especially the styloid. The greater horn of the hyoid is formed by ossification of the cartilage of the third arch.

The cartilage of the second branchial arch lies in such close proximity to the developing tonsil, that such investigators as Grünwald and Theodore believe it is quite possible for cartilage rests to be included. Indeed, Grünwald makes the statement that in some cases an outgrowth of the cartilage of the second arch actually projects into the tonsil as a support. Considering the fact that the connective tissue of the tonsil is fairly complete before the lymphoid tissue begins to develop. the exponents of the cell rest theory maintain that rests of cartilage are likely to be confined to the connective tissue. Instances are on record where rests of the first visceral arch have developed into supernumerary ears.

As for the multiplicity of nodules, Reitmann records the finding of isolated cartilage cells in the tonsil of a sixteen-year-old girl, while Reitmann and Rückert found as many as three cartilage nodules in the tonsils of the newborn.

And this answers another objection, namely, that the findings have all been in the case of adults, whose tonsils gave evidence of past or present inflammation. In 1904 Rückert investigated twenty-four pairs of tonsils from infants varying in age from eight months of fetal life (miscarriages) to one year. Out of the forty-eight tonsils, seventeen showed the presence of cartilage, within the connective tissue of the capsule, near the mucous glands. In only one case was there any evidence of cartilage to the naked eye. In each of two cases two nodules were found. The cartilage was embryonic in character, rich in cells, and poor in ground substance. Often there were two cells in a space, indicating rapid growth. In every case there was a definite perichondrium.

The same year Lubarsch examined twenty-three tonsils from twelve cases ranging in age from fetuses of six months to nurslings of seven and one-half months. In a fetus of eight months he found a cartilage nodule containing fairly large cells and elastic fibers, and surrounded by connective tissue.

Reitmann also found cartilage in a fifty-one millimeter embryo, and in several very young children, in whom the tonsils were otherwise unchanged, as did Anselmi, whose description of his cases we have been unable to obtain.

One of the strongest arguments of those holding the metaplasia theory is the fact that most cases show evidence of past or present inflammation. Walsham, Wingrave, Lubarsch (fifty-nine out of sixty-five cases, including one two-and-one-half-month-old infant), Rückert Reichert, Nösske, Halkin, Theodore, and Cary all worked with cases of that kind. But the opponents of the theory point to the fact that in case of the fetuses and newborn there has been little opportunity for inflammation, and that some of the tonsils of adults show no evidence of inflammation whatsoever.

As for the fact that the phenomenon frequently appears late in life, the adherents of the embryonic theory point to the fact that embryonic cells often lie dormant until late in life, as for instance, the germinal cells.

The histologic findings, such as the relationship between bone and cartilage and the surrounding connective tissue, and of the bone and cartilage to each other, have already been discussed. Those who hold the process to be metaplastic, regard the frequency with which the nodules are in intimate contact with the connective tissue as evidence that they originated from it. Those who hold the embryonic theory ignore that fact, but point out carefully the instances in which there is a real or apparent capsule surrounding the bone nodules, in which ossification seems to have nothing to do with the surrounding tissue.

Zeigler, Lubarsch, Nösske and Cary all regard either theory alone as insufficient. Neither covers all the cases, and in some it is probable that both factors were at work at once. That is, the embryonal rests may lie dormant until abnormal conditions in the tonsils rouse them to activity.

The conditions where the hyoid or other bones become sufficiently enlongated to encroach upon the tonsil come outside the pale of this discussion. They form a class by themselves. But Deichert and others hold that, as they originate from the second branchial arch, they indicate an abnormal activity of the arch.

REPORT OF OUR CASE.

The specimen was sent by Dr. F. W. Bailey of Cedar Rapids to Dr. L. W. Dean, by whom it was referred to us.

Clinical History.—Male, age fifty years, physician. Has had most of the common diseases of childhood. Since childhood has had several acute attacks of tonsillitis every year until a few years ago, and mild attacks in recent years. During childhood the tonsils were enlarged, but at the time of enucleation they were so shrunken that they were scarcely visible in the throat. General health good, except for arthritis deformans, as evidenced by pain, stiffness, slight redness, the presence of Heberden's nodes on the knuckles of both hands, which started eight weeks before the tonsillectomy was performed. Has never had rheumatism. The throat was granular. The patient is not a user of alcohol, and drinks tea and coffee moderately.

The tonsils were found to be firmly fixed in the fossæ, and removed with difficulty. No bone tissue was cut at the time of operation. There was considerable hemorrhage, which was, however, readily controlled by the use of hemostats. Recovery was uneventful. The presence of bone was suspected after palpation of the enucleated tonsils.

Gross Pathology.—Both tonsils are about the same size. After being in formaldehyd fixing solution for several weeks, each measured twenty-three millimeters in length, and nine millimeters from surface to base. In width, one was sixteen millimeters and the other eighteen millimeters. Both were removed with the fibrous connective tissue capsules intact. The surface of one is fairly smooth, that of the other somewhat irregular, due to the opening of rather deep crypts. Both tonsils are fairly firm, and each contains a substance near its base of bony hardness and rigidity. Nowhere could bony or other calcareous material be felt on the surface. Bone was reached only on cutting the tonsil and was easily cut through by the knife. The lymphoid tissue of both tonsils was of a normal light grayish color. In each, extending beneath the capsule, was a thin plate of bone, light colored, and easily felt by means of forceps. In one, in which it extends for sixteen millimeters in the capsule at the base, it projects as a thin plate toward the surface for a distance of six millimeters. In thickness it varies from one-half to two millimeters. (See Figure 3.) At the ends of the projections is light colored tissue, of cartilaginous consistency, and devoid of calcareous matter. In the other tonsil the plate measures eleven millimeters in length and one millimeter in thickness. As far as could be determined by palpation, the plates extend laterally across the base of each tonsil for a distance of eight millimeters. In each case the cut surface of the tonsil shows the calcareous material to be in the form of one large piece and several small islands, but we believe the apparent islands are irregular projections of the main mass.

Microscopic Pathology.—A section through a decalcified piece of one of the tonsils shows the presence of seven pieces of bone. (See Figure 4.) The larger pieces are elongated in the form of plates corresponding to the calcareous plates described in the gross specimen. The bony material contains several large openings, some of which are lined by endothelial cells, and contain red blood corpuscles and a substance resembling fat in appearance. The bone formation appears in the form of concentric whorls arranged about the larger openings. In the bony tissue proper are a large number of small spaces containing cells, with canaliculi radiating from them. The whole appearance presents the arrangement of the Haversian system of true bone. There is a sharp line of demarcation between the bone and the surrounding connective tissue. A section was made through a portion of the tonsil containing a small, firm nodule, presenting the appearance of cartilage and offering the same kind of resistance to the knife. It was not decalcified, yet it was easily sectioned by the microtome knife. It contains concentric lamellæ, bone-like cells, and canaliculi, but no trace of cartilage. The intercellular substance is rather homogeneous, with a small amount of granular calcareous deposit. It is well differentiated from the surrounding tissue. Around it, however, one can see a gradual transition from connective tissue cells to rather large, round cells, with a large amount of cytoplasm, suggesting osteoblasts. The connective tissue here is distinctly increased in amount, invading the lymphoid tissue in some places, and appearing as a broad, dense band in others. Where the increase in connective tissue is most marked, there is a large amount of adipose tissue and some areas of hyaline degeneration. The lymphocytes are closely crowded, indicating a hyperplasia. The blood vessels have thickened walls and little blood. There are several small, old hemorrhages, characterized by the presence of broken-down red blood corpuscles and hematogenous pigment. The germinal centers are poorly defined.

We also made one section of lymphoid tissue furthest away from the bone. The germinal centers here are well defined, many cells showing mitotic figures, as under normal conditions. The blood vessels are rather distended, especially the capillaries. The connective tissue appears normal. The surface is covered with a rather thin layer of squamous epithelium.

DEDUCTIONS FROM OUR CASE.

When we analyze our case, we are forced to conclude that it is the result of bone metaplasia, rather than of the displacement of cell rests from the cartilage of a branchial arch, and this in spite of the fact that some of the conditions are similar to those which supplied others with arguments for the cell rest theory.

1. From the clinical history we learn that the patient was a sufferer of tonsillitis almost all his life. Microscopically, we find an increase of connective tissue in the neighborhood of the bone.

2. The shape of the bone, which is that of a long thin plate, with extensions into the connective tissue septæ, suggests a definite activity on the part of the connective tissue involved, rather than a development from a cell rest, which would have been more likely to take the form of a fairly regular growth, tending to be spherical in shape.

3. There is no evidence of cartilage. On the contrary, we find a portion of the bone so soft that it could be cut by the microtome knife without decalcification, yet having the appearance of true bone. This indicates that the young bone was not preceded by cartilage. A similar formation was reported by Harvey[23] in a case of experimentally induced bone formation

in an artery of a dog, a case which was one of undoubted metaplasia.

The exponents of the branchial arch theory take the presence of a periosteum to indicate cell rests as the origin of the bone. But our periosteum is in intimate connection with the surrounding connective tissue. Is it not possible that metaplasia here took the form of transforming connective tissue into periosteum, and that the latter gave rise to membrane bone? Normal periosteum is formed by a differentiation of connective tissue cells into osteoblasts.

It seems to us that the argument that the connective tissue of the tonsil develops previous to the lymphoid tissue does not imply that rests from the second branchial arch would necessarily be confined to the connective tissue. No less an authority than Hammar[21] maintains that the lymphoid tissue probably forms from the connective tissue cells. If that is the case, it is quite possible that the lymphoid tissue would, at least in some instances, include some of the cartilage cells so common in the connective tissue. But such case has never yet been reported. The limitation of cartilage and bone to the connective tissue only, therefore, probably indicates an activity peculiar to that tissue.

We do not mean to imply that all cases are the result of metaplasia, but we do maintain that the evidence held by many of the investigators as practically conclusive proof of the cell rest origin of bone and cartilage in the tonsil is not at all above criticism.

SUMMARY AND CONCLUSIONS.

1. The first report of a case of bone and cartilage in the tonsil was made by Orth in 1893. Since that time several hundred cases have been reported.

2. Bone and cartilage in the tonsil have been found in the following forms:

   a. The elongation of the hyoid bone or styloid process of the temporal bone in such a manner as to extend into the tonsil.

   b. Bony plates or nodules and cartilaginous nodules.

3. Interest in the phenomenon centers chiefly around the problem of the origin of those bony and cartilaginous masses

in the tonsils, not connected with the skeleton. Investigators have taken sides on two points of view.

a. One group holds that they are a result of metaplasia following inflammation in the tonsil.

b. The other group maintains they are the result of cartilage cell rests from the second branchial arch just below which the tonsil has its origin.

4. It seems evident that a large number of instances can best be explained by the cell rest theory.

5. Our case, however, seems to fall into the class of bone metaplasia, in spite of the fact that it contains some features which have been used as arguments for the cell rest theory.

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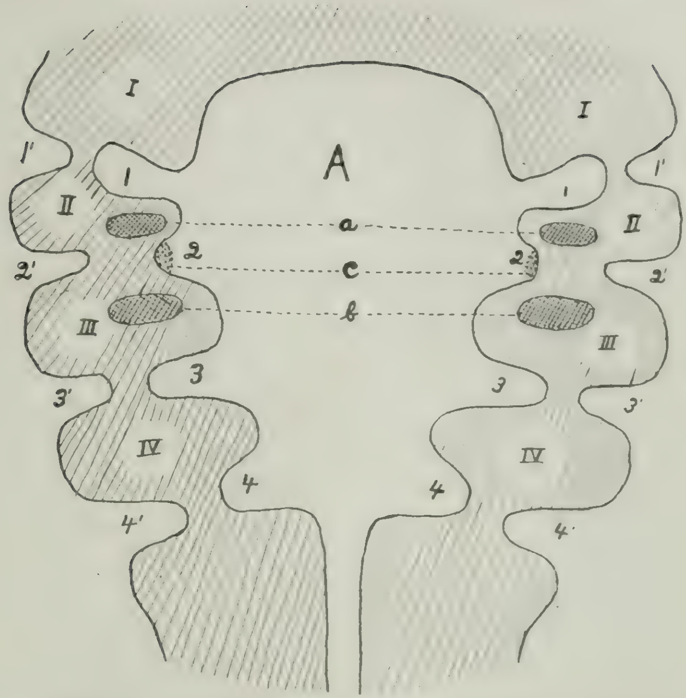

Figure 1.—Diagram showing the origin of the tonsil and its relationship to the adjacent embryonic structures.

I, II, III, IV.—The four branchial arches respectively.
1, 2, 3, 4.—The four pharyngeal pouches respectively.
1', 2', 3', 4'.—The four branchial clefts respectively.
A, pharynx; a, cartilage bar in second branchial arch; b., cartilage bar in third branchial arch; c, developing tonsil.

Note especially the proximity of the developing tonsil to the cartilage bar of the second branchial arch. Rests of cartilage may be misplaced from the latter into the tonsil. From this cartilage is formed, the styloid process of the temporal bone, the lesser cornua of the hyoid bone and the stylohyloid ligament connecting these two bony processes.

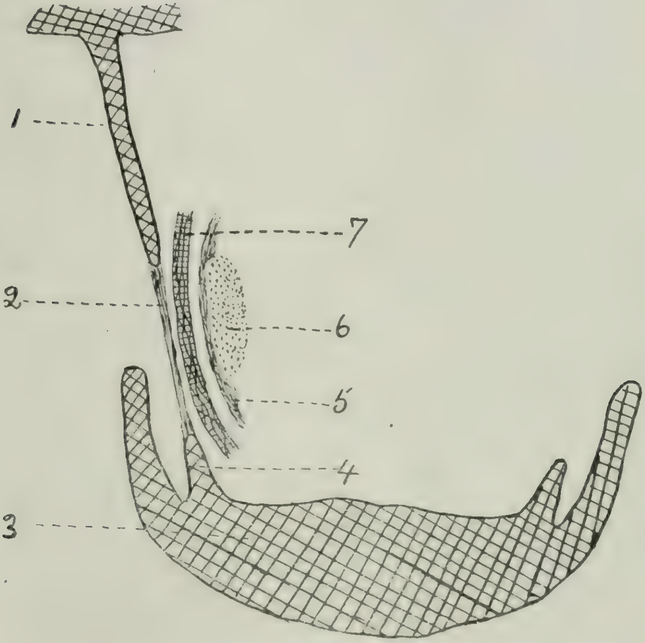

Figure 2.—Diagram showing the relation of the tonsil to surrounding tissue—more especially to the bony processes noted.

1, Styloid process of the temporal bone; 2, stylohyoid ligament; 3, hyoid bone; 4, lesser cornua of the hyoid bone; 5, capsule of the tonsil; 6, tonsil; 7, constrictor muscle of the pharynx.

There are reports of several cases in which either the styloid process of the temporal bone or the lesser cornua of the hyoid bone projected into the tonsil. Such represents one of the three explanations for the presence of bone in the tonsil.

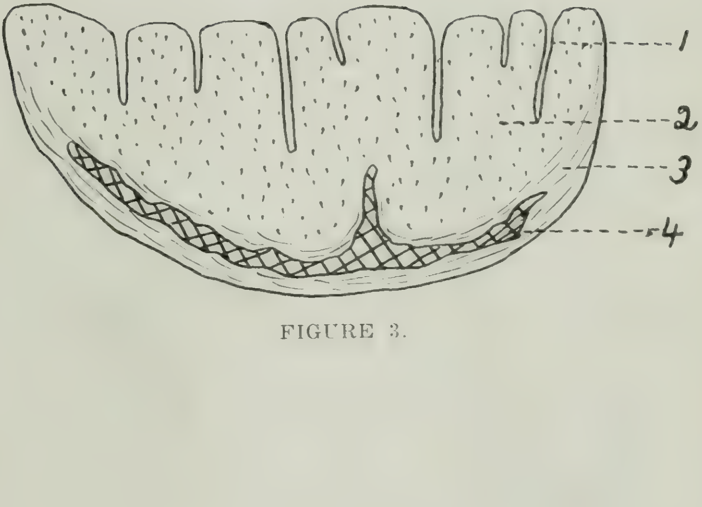

FIGURE 3.

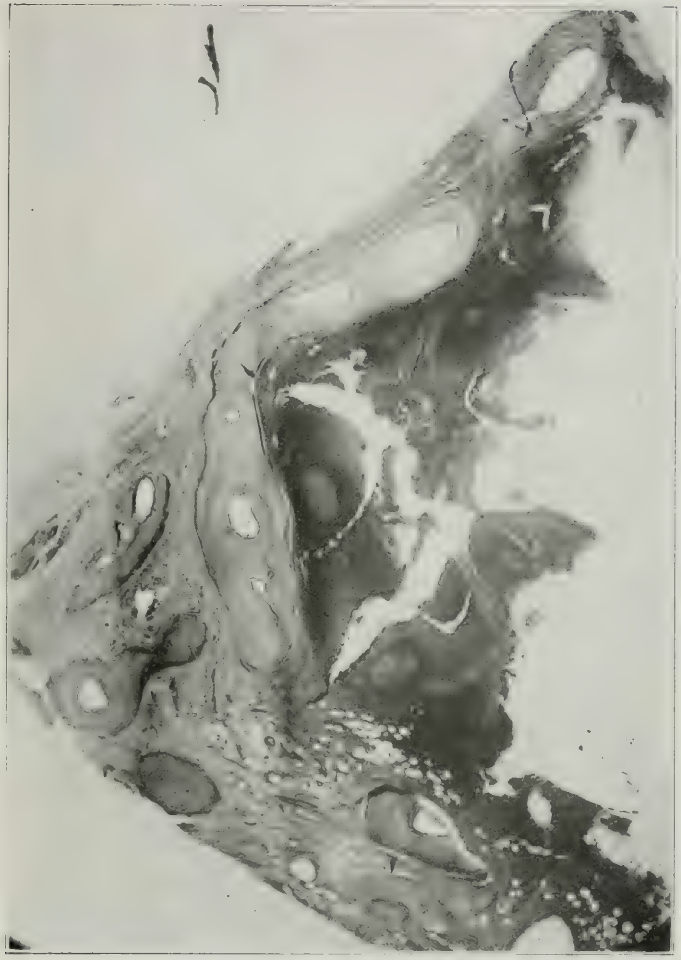

FIGURE 4.

Section through tonsil. Decalcified. Stained with hematoxylin and eosin. Low power. Shows the presence of bone (seven areas) in the capsule of the tonsil. Note that the bone areas are well defined from the surrounding tissue. The lamellæ, bone cells and large central spaces containing fat cells are distinctly noticeable.

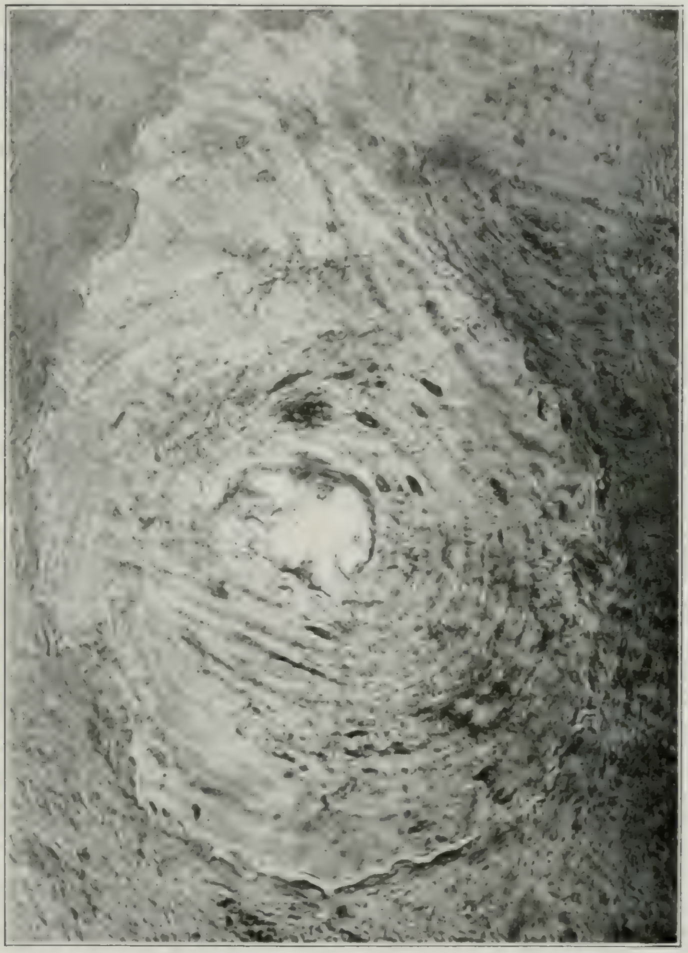

FIGURE 5.

Section through area of bony nodule in tonsil which contained but little calcareous material. Cut without decalcification. Stained with hematoxylin and eosin. High power. Note the central cavity which contains fat cells; the lacunæ with the enclosed bone cells, and canaliculi radiating out from them; also the layer of rather large cells at the periphery of the bony area which probably represents a periosteum.

LV.

DOUBLE OTITIS MEDIA.—SUBMILIARY PULMONARY TUBERCULOSIS.*

By Emil Amberg, M. D.,

Michigan.

Miss E. W. R., nineteen years old, consulted me December 19, 1916, on account of difficulty in hearing in her right ear which interfered with her work as bookkeeper. The patient, an anemic girl, was very anxious not to miss any work, and not to lose much time. She said she had been hard of hearing in her left ear for four months, and that recently she became hard of hearing in her right ear. The right ear became much worse on the day before yesterday. The right drum membrane was injected, and when the right ear was syringed the fluid went into the throat.

On the left side there was a chronic middle ear suppuration lasting for several months. If I remember correctly, the discharge was offensive and very scanty. Patient has been hoarse for a long time.

On the next day when patient came again, I heard a slight hacking cough. The temperature by mouth was 101.2. The general appearance of the patient did not correspond to the picture of an otitis media and its complications. I therefore, asked the patient to have her chest rayed.

Report on roentgenographic examination, December 20, 1916 (see Figure No. 1).

"(Drs. Hickey and Evans.) Stereoscopic plates were made of the chest. We would report that there is a very definite fine infiltration scattered quite universally through both the right and left lung. This assumes the form of very small snowflake-like deposits. This we think, should be classed as a diffuse, submiliary tuberculosis."

(Signed) W. A. Evans.

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*Read before the American Otological Association, May, 1917.

I advised the patient's mother to have the girl enter Harper Hospital, which she did December 22nd.

From the Harper Hospital records, the following may be mentioned:

December 28, 1916. Wassermann negative.

December 29, 1916. Report of roentgenologist. (See Figures 2 and 3.) "Plates were made of the right and left mastoid. The right mastoid shows rather a large, crisp, pneumatic mastoid, some of the cells of which are quite large and fairly well spaced. The left mastoid does not appear nearly as crisped; the outlines are indistinct. We would think that there was an exudate constituting probably a first degree mastoid."

(Signed) WM. A. EVANS.

December 22nd, 1:15 p. m. Temperature, 99; pulse, 96; respiration, 20. Patient complained of sharp pain in head; ice bag applied to head. Examination of chest and back made by Dr. Wehenkel. 7:30 p. m. Sodium salicylate, gr. V; phenacetine, gr. II; codein sulphat, gr. ¼. 9 p. m., feeling much better; headache much relieved. Unable to obtain specimen of sputum.

December 23rd, 8 a. m., incision of right drum membrane. 2 p. m., complained of severe pain in head. Medication ordered by Dr. Wehenkel, containing sodium salicylate, phenacetine, codein phosphate, camphor monobromid. Similar medication was given on subsequent days until about December 31st, when sodium salicylate and phenacetine were discontinued. Aspirin, grains V, was given January 3, 1917, once; January 4th, twice; January 5th, once. These medications, among others, are mentioned only because the temperature and pulse may have been influenced to some extent by the same.

The nights, up to January 4th, were as a rule comfortable; from January 4th to 5th, the patient slept very little; from January 6th to 7th, patient was very restless; whereas from January 7th to 8th, patient had a very good night, but not from January 8th to 9th.

January 5th, patient unable to walk, left hand twitching. At 8 a. m., pulse weak and irregular; at 12 noon, patient cyanosed, respiration heavy. January 8th, 7:40 p. m., respiration shallow; at 8 p. m., patient was unconscious, and passed away at 9:10 p. m.

Dr. A. M. Wehenkel, who occupies himself particularly with tuberculosis, kindly examined the patient, prescribed for her and reports as follows:

Family History.—I did not learn anything about her family.

Past History.—Three years ago she had an attack of coughing—began to lose weight—had lost considerable weight in a short time; had frequent attacks of cold. It was then called "bronchitis," the cough persisting. Had several attacks of pleurisy during past three years. She temporarily recovered until last fall when took "fresh cold," the cough was renewed; she began to lose weight, P. M. fatigue set in, pain in right side aggravated by long breaths, blood tinged sputum occasionally; night sweats; fever in evenings, an incessant cough set in. Some nights she would cough all night without any sleep.

Meningeal Symptoms.—She developed a headache which was gradual in onset, and progressive in severity; she vomited occasionally, not associated with nausea; she was constipated constantly. After four or five days her eyes became somewhat fixed—double vision. She began to have pain in her spine with a little stiffness in her neck. When I saw her last there was no Kernig's sign; temperature rise in evening.

Physical Findings.—Larynx, mouth and nose were negative; the expansion of chest was diminished on both sides, but more lagging on the right side; no depressions. On palpation the right side showed an increased fremitus, extending over the upper two lobes, quite marked posteriorly between 4th and 6th interspaces. On percussion both apices were retracted, more marked on right; percussion note dull over middle lobe posteriorly; the resonant area was quite high on right side posteriorly.

Auscultation.—Breath sounds over right apex were diminished with high pitch and prolonged expiration to fourth rib anteriorly; posteriorly to about seventh. Harsh respiration over upper left lobe, scattered crackling rales over entire right base of lung; vocal fremitus much increased over middle lobe posteriorly; fine rales were present over left apex. In conclusion, I would say that she had a bilateral infection. The condition was a fibroid tuberculosis involving both sides for some time quiescent, and which had become very active, undoubtedly taking on a miliary form with an extension to the meninges. Owing to the very high temperature, it is possible that she had an accompanying secondary infection.

Additional Remarks.—While writing the paper, I learned from a friend of hers that about two weeks before going to the hospital friends met the patient at a party where she complained of pain in her ear (which?), and that a physician told her that she needed rest more than anything else, that other friends commented on her white complexion, with a bright red spot on each cheek. The same friend visited her in the hospital two or three days before she died. She looked a long time at the friend and said: "Why, is that Rena? I can hardly see you."

The mother says that the patient always complained of headache, and that she had irregular menses. In May started with a severe headache; she never complained to have earache, always headache, except when she was in the hospital when the right drum membrane was incised.

The mother says that during the last week the patient saw double with and without her spectacles. An aunt was sitting on a chair and the patient thought the aunt was sitting on the floor. Patient could not see well without glasses, but she did not have double vision before she entered the hospital.

A few days before patient came to me, she said: "After Christmas I am going to take an inventory of myself. I have not got a well spot on me, I ache all over." She thought the office would not get along if she were not there. Twice she was brought home after having fainted; once after leaving the street car, once in the office. The mother also reports that the daughter never told her anything about the left ear for fear she would send her to a physician, but the mother learned from a friend of her daughter, that the patient complained for a long time, now and then, of quite severe pain in the left ear, but that the ear did not discharge. The patient had also complained that the whole left side of the body felt numb.

SUMMARY.

The patient consulted me on account of a disturbance in her right ear, which interfered with her work. The patient was entirely unaware of the seriousness of her general condition, even to the end. (Her employer told me that he had warned the patient for some time to take care of herself.) The finding in her lungs at once reversed the whole viewpoint on account of the bad prognosis. On account of the miserable condition of the patient some of the routine examinations, which would have been made, were not made. The lumbar puncture might have been the most important one from a diagnostic standpoint.

The symptoms on the part of the brain and its coverings were characteristic for meningitis. A gradually developing cloudiness of the mind, more or less severe remitting headache, constipation; on December 29th, tenderness on pressure, not so much over left mastoid as in surrounding tissue (probably part of a general hyperesthesia); January 4th, creeping pain in neck (relieved by hot water bag); January 2nd, a complaint that she can see, but cannot make out anything.

It must be considered what importance must be attributed to the disturbance in the left tympanic cavity and mastoid process; also, whether the meningitis resulted therefrom, or, whether the tubercular trouble was aggravated by the presence of this focus; or, whether there existed no relation between the two conditions.

Concerning a possible exploratory operation on the left mastoid (see also X-ray report), there may perhaps be a difference of opinion. Dr. D. P. Mayhew, in a paper before the Wayne County Medical Society, Detroit, May 14, 1917, reported a number of operations, including mastoid operations, which have successfully been performed in Colorado on patients suffering from tuberculosis. Whether such a grave general condition as that found in our patient would have permitted an operation on the left side, even if the same had been much more clearly indicated, is very doubtful, especially at this stage of the general disease.

An autopsy was not performed.

LVI.

TUBERCULAR MASTOIDITIS—RADICAL OPERATION UNDER COCAIN ANESTHESIA.*

By Harold Hays, M. D.,

New York.

About two years ago, Mr. S., aged twenty-four years, came to consult me about his deafness and sharp pain in the right ear. His history showed that he had had a tuberculosis of the lungs which at that time was in a quiescent state. He had had a radical mastoid operation performed on the left ear and mastoid four years before. This had never healed properly, with the result that there was a large opening into the dura which was constantly suppurating.

Examination of the right ear showed granulations in the middle ear, entire destruction of drum, and the discharge of cheesy-like pus. Culture from this ear and from the sinus behind the left ear showed numerous tubercle bacilli. He could hear neither watch nor tuning fork at either ear.

During that first winter I had him under observation two or three times a week, during which time I attempted every known means of clearing up the condition.

As he did not improve, I advised a radical mastoid operation.

The question of anesthesia was an important one. Here was a patient with a quiescent tuberculous process in his lungs, a patient whose general physical condition was far below par. Ether anesthesia per oram would possibly light up his lung condition, so I determined to operate upon him under local anesthesia.

On the day of the operation at ten o'clock in the morning he was given a hypodermic injection of morphin grain one-fourth, atropin grain one-one-hundredth. Two hours later a second injection of like amount was given. At two o'clock he was brought to the operating room. As there were no signs of opium poisoning, another injection of morphin grain one-eighth was given. The operative field was prepared in the usual manner. A solution of equal parts of ten per cent cocain with equal parts of adrenalin having been prepared, an injection of this solution along the line of the proposed incision was made, going well into the surrounding tissues, forward to the lobe of the ear, upward to the zygoma, and backward to the occipital bone. An attempt was made to have the injections reach down to the periosteum. This is not a difficult matter in this region, as the tissues are not very thick.

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*Read before the New York Academy of Medicine, Section on Otology, October 13, 1916.

The semilunar incision was made throughout its whole extent down to the periosteum. A longer needle was attached to the hypodermic syringe and an injection made under the periosteum of a one-half of one per cent cocain solution with equal parts of adrenalin (Shleich infiltration). Two to three drams of this solution were injected, care being taken that some of it reached the periosteum below the tip and anterior to the bony canal. It was spread through the tissues posteriorly and above until an area of at least three to four square inches was infiltrated.

The periosteum was then incised and scraped back with a rasp and the retractors were inserted.

After the bone was well exposed the mastoid cavity was uncapped with a chisel. The patient complained of no pain at any time while I was working in the mastoid cavity. The operation was a Schwartze-Stacke. The cavity was extremely large, the bone extremely hard, the tip entirely destroyed so that it had to be removed in its entirety. When the patient was asked to describe his sensations, he stated that he felt no pain, only the apprehension of pain, similar to what he would feel in the dentist's chair. We are wont to feel that in the mastoid operation under ether anesthesia, the constant chiseling and using of rongeurs produces a slight concussion of the brain, which accounts for the lessened amount of ether we have to give. After this experience I am inclined to think that this idea of ours is visionary. The constant pounding of the bone produced no marked change in the patient's mastoid condition. In fact, he did not seem to mind it at all.

The mastoid cavity was thoroughly cleaned out without any trouble. The bony canal wall and the bridge over the antrum were taken down without any pain. However, when the sensitive mucous membrane of the middle ear was reached, trouble commenced. It had been impossible thus far to cocainize this membrane. Here was a mass of sensitive granulation tissue. In order to overcome this sensitiveness a four per cent cocain solution was injected along the posterior canal wall, into the granulations themselves and deep down into the eustachian tube. Not all sensitiveness could be overcome, but enough to proceed with the operation with little trouble. The mass of granulations were removed with a curette until I came to a thicker tougher mass, apparently attached to the internal wall of the middle ear. I hesitated to remove this, as I realized that the facial nerve was either in it or close by. But the mass was infected and had to be removed. I grasped it with forceps and snipped it off with a small scissors. At once the patient exclaimed: "Doctor, you must have hit my facial nerve. The right side of my face is paralyzed." Such was the fact.

The eustachian tube was curetted and a skin flap for a new canal made without any trouble. The making of the skin flap was facilitated by the lack of bleeding, due to the blocking off of the vessels by the cocain solution. When one realizes how much bleeding there usually is, he can appreciate of how much value this ischemia was. After making the artificial canal the posterior wound was closed with tendon sutures.

Three hours after the operation the patient sat up in bed to eat his supper. Having had a radical mastoid operation performed on the other side under ether anesthesia, he was in a position to make comparisons. He was most enthusiastically in favor of local anesthesia. Not only had the general reaction been severe at the time of the former operation, but his wound pained him a great deal. In this instance the pain was practically nil. At the end of the week he left the hospital with his wound in excellent condition. The facial paralysis began to improve, so apparently the nerve was not completely severed.

A few months ago I lost track of the patient. I had hoped to present him at this meeting. But I received a letter a short time ago stating that he had died at Bellevue Hospital during the summer from a tubercular meningitis. Autopsy showed a direct extension of tuberculosis from the suppurating cavity behind the left ear to the dura.

The practical points to be deduced from this case are the following:

1. That the radical mastoid operation can be done under local anesthesia without any pain.
2. That the superficial scalp tissues and periosteum are sensitive, but that bone has absolutely no sensation.
3. That the mucosa of the middle ear is extremely sensitive and must be separately cocainized.
4. That irritation or destruction of the facial nerve is immediately noticeable to the patient.
5. That the after-affects are practically nil.
6. That the end result is just as good under local as general anesthesia.

LVII.

OZENA AND ASPHYXIATING GAS—A NEW ETIOLOGIC STANDPOINT.*

By Marcel Natier, M. D.,

Paris, France.

(TRANSLATED BY EMIL MAYER, M. D., NEW YORK CITY.)

I experience a double satisfaction in again reverting to the study of this subject, to which I have many times alluded in the past. Indeed, I deem it a commanding duty, with my thoughts fixed on that gigantic struggle of the nations, which goes on unceasingly, without limitations, shaking the very fundament of the whole world from its most ancient until the present time.

Furthermore, there is a melancholy pleasure in adding a new link, quite unexpected, to the chief causes of a malady into whose intimate nature I have given much particular attention for many years, for it so happens that it is one of the glorious victims of this war whom I have to thank for furnishing me with the theme of this communication.

Here is his lamentable history:

F., born in 1891, about to finish his military service when called upon for mobilization. Father, workman, in the port of Dunkirk, sober, good health, died of anthrax. Mother, aged sixty-seven years, good health, excessively stout, no nasal affection, no consanguinity. There were six children, the oldest dying at two months. One brother had an affection of the lungs from exposure, and recovered; three sisters in good health, and the patient.

Personal History.—Nursed by his mother, leaving his native town only when he joined his regiment; had no privations. From six to thirteen years went regularly to school. Employed in a silk-yarn factory until his eighteenth year. No overwork; but constantly exposed to dust. From the eighteenth to the twentieth year ship carpenter, then soldier.

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*Read before the American Laryngological Association, May, 1917.

As a child, scholar, workman, soldier, always in good health; no venereal affections, no headaches; his digestive, circulatory and nervous systems were absolutely normal up to the time of his injuries and asphyxiation. Respiratory system entirely normal, no nasal secretion or epistaxis, no sinus disease, no adenoids or tonsils.

October 18, 1915, he received five bullet wounds in his lower limbs. On the right side a superficial wound in the hip, one below on the calf and one much deeper in front of the tendon Achilles. On the left side, wound on the anterior portion of the abdomen; immediate removal of the projectile. Wound with an orifice below the knee, and on the external border of the left hip extending to, and having its exit in, the internal part of the leg below the calf.

Asphyxiating Gas.—Bombardment with asphyxiating bombs ("Obus"). At first, owing to poor calculations as to distance, the bombs did no damage, and the odor was but dimly perceived. Subsequently, by direction of aviators, this was rectified, and the bombs reached the trenches and great ravages ensued.

While in an open field, covered by the enemy's artillery, our patient was struck by an "Obus," and asphyxiated by the gas. He was not protected by his mask, which had dried up. He fell like a log; asphyxia followed with total loss of consciousness for several hours. Eventually he regained consciousness, followed by renewed attacks of syncope.

After about thirty-six hours he was placed in a conveyance, still clad in his clothes, soiled with vomitus and a great deal of blood. Blood mixed with greenish matter was not expelled except by hiccough. No evacuations took place. Coughing and respiration were impossible without most exquisite pain and severe burning in the stomach and chest. "Every effort of the kind tears my lungs," said the patient.

October 15th, the next day after the injuries, he was transferred to Vitry le Francois, where he was obliged to remain for three weeks, owing to his febrile condition. Skin cyanotic, respiration prolonged with great agony, lasting at least a fort-night. Almost total impossibility of respiration; each attempt followed by severe stitching pains, extending from the nose to the bifurcation of the bronchi. Received special care, dressing of wounds. Several injections of morphin and of serum; inhalations of oxygen—only two of these were given.

November 11th, arrival in Paris at base hospital. Daily injections of normal serum and of morphin four times in the day; inhalations of oxygen; mentholated vaselin for the nose obstruction.

For the five months following the injuries there was much difficulty of respiration. For at least two months, inhalations of oxygen were repeated three to four times a day, ten to fifteen minutes each time, and though faintings often occurred after inhalations, they were followed by amelioration of symptoms.

Partaking of food by the mouth was absolutely impossible for the first six months, terrible burning in the throat followed attempts at swallowing, then dreadful vomiting from an intolerant stomach, even for milk and ices; so for two months irrigations of two hundred and fifty grams of normal serum were daily performed.

There was a permanent febrile condition, total insomnia, intense feebleness, wasting cachexia.

March 18th admitted to the Saltpétrière, service of Professor Dejerine. There was feebleness of external extremities, intense headaches, respiratory troubles; ozena and vomiting reappeared for a month following electrical applications.

Examination—Inferior Members.—All possible movements greatly reduced. Feebleness not marked on the left, while emaciation was greatest on the right. No hypotonia, no contractures, reflexes normal. Contractions of muscles normal.

Upper Extremities.—Muscular power slow and weak, reflexes normal. Respiration rapid and irregular, 120; heart and lungs normal; pulse, 104.

August 4th, absolute uselessness of lower extremities; respiratory troubles diminished.

August 22nd, strong faradic current. Galvanic current of thirty milliamperes, followed by slight voluntary movement of great toes. Attempts to move the patient followed by tremor of hips. Some muscular contracture of hips, but no movement.

October 25th, I first examined him. The two alæ, more particularly the right, were strongly collapsed, remaining in contact with the septum; otherwise very mobile. In the left nasal fossa slight atrophy of the middle and inferior turbinates. Deviation of the right bony and cartilaginous septum; concavity on the left. Mucosa dry. Right side of nose, same condition as on the left, except for greater space because of the deviation. No trace of sinuitis. There was a catarrhal state of the vocal cords. Mucous membrane of the nasal fossa markedly anemic; general sensibility normal; considerable diminution of odor; mouth perfect, dentition always; taste diminished, pharynx normal.

Vision.—Bad on the right, where nothing can be distinguished; left eye closed.

Skin.—Normal, no excessive perspiration hands or feet; excessive perspiration in arm pits.

Digestive System.—For a month after admission to the Salpétrière has been enabled to partake of food by the mouth, although he still has eructation and weight in stomach, with some vomiting one-quarter of an hour after ingesting food. Increase in weight and disappearance of constant sensation of vertigo.

From the beginning, intestinal evacuations only once in three days. Purgations ineffectual. No desire for evacuation and inability to pass gases from rectum. Enemata were resorted to; the patient did not even feel their being used.

Nasal Respiration.—Complete stenosis from collapse of alæ from the very first. If asleep with mouth closed, he is promptly awakened by suffocation, so he became accustomed to place the ends of matches in his nostrils each night regularly to keep them apart. He was a constant mouth breather. With the mouth closed, respiration became jerky, waking or sleeping.

Ozena.—Three to four months after the injury, and for the first time, there was a manifest alteration of the nasal secretion; yellowish at first, progressively becoming thicker, and about one month later, crusty and greenish.

Expulsion of bloody crust only after great efforts, requiring hundreds of attempts for an hour; then they would eventually become dislodged.

Three weeks after this he became aware of the bad odor of these crusts by the complaint of a comrade, who repeatedly asked him if he had not "eaten cheese." It so happens that he never partook of cheese, hence his attention was directed to his ozena, although there was at this time a considerable diminution of the odor.

No olfactory sensation was awakened by inhaling eau de cologne, which produced burning sensations of the nasal mucosa like unto that occasioned from holding a lighted match close to the nose. At the same time there was burning of the eyes with lacrimation, especially the right eye.

Progressive increase of ozena; more frequent use of handkerchiefs; more frequent headaches, especially temporal. The tongue is always burning, with recurrent eruptions.

Treatment of Ozena.—In February, at the first examination at the Salpétrière, I advised regular insufflations of warm salt water, which, in spite of the nasal pain thereby occasioned, was followed by amelioration. There was a diminution of the amount and quantity of the crusts, which were now changed to smaller bloody "leaflets" or "balls."

January 10, 1917, an abundance and the same fetor of nasal secretion; crusts formed, entered the mouth, and, the patient being unable to expel them, were swallowed. For two weeks cessation of nasal irrigations because of severe pains they caused in the nose, with a resulting increase of nasal conditions because of drying of crusts. In spite of this, respirations seemed easier; need still to maintain nostrils artificially spread or wakening with suffocation at night. More pulling sensation in the chest. Respiration decreased to sixty.

Special Alimentation.—Two liters of milk daily, four eggs and soft foods. Meat in small amounts—and, still more remarkable, veal alone was borne. All other meats, ragouts, or sauces with onion, caused a disturbance in the stomach soon after their ingestion; hypersecretion as of boiling water, very acid; the tongue became as on fire and covered by numerous white elevations. The same fiery sensation of the lips, accompanied by a more or less confluent eruption.

The very hard masses were expulsed every two or three days at the cost of pain and agony each time, sufficient to bring out tears.

Spontaneous evacuations returned for the past month, since when gases passed freely from the rectum, and sensation returned to his hips. Enemata produced pain, and rather than submit to their administration, preferred to make efforts to evacuate his bowels by the hour.

Inferior Extremities.—Sensation, entirely absent following injury, shows a tendency toward return since about the past two months, plainly limited on each side to two-thirds of the upper part of the hip. At the junction of one-third of the inferior portion there is a ring surrounding the member; palpation on the surface produces a sensation of pricking instead of the simple sensation of contact. It seems as if the hip were attached by a cord that was being tightened. Walking always impossible, or with slight progress. Attempting to walk, the legs were projected in advance. This inability could in nowise be imputed to any unwillingness on the part of the patient, who, receiving no visitors, was actually grieved to remain fixed in one place, and was very happy to be able to visit his comrades.

Vision stationary.

March, 1917, though slowly, the ozena is improved with the general improvement. (This observation is of interest from several points.) The affection has shown an exceptional if not unique course.

Before the injury, not the slightest pathologic condition of any susceptibility to predispose to an offensive nose. This young man in the state of blooming good health, now bore every sign of that offensive condition.

We are justified, then, in directly tracing the causal factors to the asphyxiating gas and the subsequent functional disorders, an ozena purely accidental and at the same time so rapid in its existence.

No less curious is the pathogeny.

Above all, we note it often to occur after luetic affections. Bearing in mind my studies of the question, and which I do not retract, I have not changed my original views relative to the origin and development of ozena. To those who have forgotten, or who have not been informed of my views, it will suffice to state briefly that in my opinion ozena is not a true morbid entity, an idiopathic or real, but a unique and always a symptomatic expression of a profound localized disturbance of the general health.

Now, in what manner does this recorded case confirm and illustrate these deductions? That is what I shall now attempt to demonstrate.

Ordinarily ozena is considered inseparable from old and far advanced atrophic rhinitis. The disagreeable symptoms I have already previously stated are the ultimate end of a disorganization, slow and profound, of various constitutional mucosa elements—blood vessels, nerves, glands, muscles, cartilage, and even bone of the nasal fossa. Similar disorganization results in a physiologic change of unusual severity of variable causes, going back often to the first years of life. That is the general rule. We must allow for exceptions. The case here recorded belongs to the former class. The atrophy is simply developed. On the contrary, on the other hand, the ozena is of recent date, is excessively pronounced, and follows almost suddenly in an adult subject. Where is the contradiction? Only in appearance; the difference is in the cause, and the affection is the same.

In my patient the determining cause was at the time sudden and violent; the effect cruel and deplorable. That the unfortunate man at the arrival of succor did not succumb immediately or in the general delay, is marvelous. How many others so stricken would have been able to return to normal equilibrium except at the price of most laborious exertions?

A young colleague, recently seen, tells me that he has never seen a case of gas asphyxiation that did not produce pulmonary tuberculosis. An experience of thousands of others. Less terrible proves to be that of my soldier. His unfortunate history in that sad physiologic loss substantiates my views, in which I persist more than ever as to the causes of all ozena.

Here viewing its nature and course, the first cause determined, the stages were particularly rapid. A wealth of evidence that the disturbance so marked of the respiratory and digestive tract proves my contention that ozena is nothing but the manifestation consummated and localized in a special part of the body, an anatomic physiologic evidence of the profound disturbance of the mucous membrane of the economy of the whole body.

Prognosis.—There is not the slightest doubt. It can only be favorable. No irreparable lesions, only functional disturbance. They become less progressively and follow the general improvement. We may count upon radical cure in accord with the latter condition.

Conclusions.—An ozena plainly of sudden origin in an adult after grave functional traumatism, a great wound, following asphyxiating gas. The rapidity of the progress is easily explained by the disorganization, at once profound and general, of the surface affected.

Once again it is verified, and supports the assertion that ozena is but a consequence, distant more often, but also under color of exceptional events of the physiologic calamity. The cause of the latter is of less importance. Its intensity merits consideration.

This truth admitted and its verity visibly impresses me. We are compelled to discard, as I have always done in my studies of the subject, the various theories, more or less fantastic, and in particular the microbic theory, invoked to explain the production of ozena.

Logically the clinical appearance, and that only, is the guide for our information that we must be instructed in the real cause of the grievous affection which is the object of this present communication.

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LVIII.

CARCINOMA OF THE EXTERNAL EAR WITH THE REPORT OF A CASE.

By Frank Warner, M. D.,

Columbus.

Carcinoma of the external ear is of such rarity that the encountering of a single case is of sufficient importance to report. It is with this thought in mind that I present a case operated on by me over eight months ago. With a report of this case, a review of the literature is presented.

C. F., male, aged seventy-seven, first noticed a small ulcer on the helix of the left ear one month before the operation, which was on December 11, 1916. Mindful of the fact that many times a carcinoma on and above the upper lip is of the hair-matrix variety, a careful examination was made for any of the characteristic signs or symptoms of this disease, as its slowly growing nature, raised, pearly edges and superficial, though broadly infiltrating behavior, but none were found. Taking into consideration the age of the patient, seventy-seven, and the fact that he had been employed recently in work that kept him continually exposed to the sun's rays, a new experience for him, producing a sailor's skin, and the general appearance of the ulceration, the conclusion was drawn that the disease was probably carcinoma of the external ear, situated on the helix, and superinduced by the irritating influence of the sun's rays. While there was no glandular involvment, the skin was adherent to the underlying cartilage. The growth was freely removed, taking in all appearance of diseased tissue, necessitating the removal of some of the surrounding healthy skin and some of the underlying cartilage to which the ulcerating mass was attached. The histologic examination of the tissue removed showed it to be a carcinoma of the squamous celled type. While the epithelial cells had freely broken through into the subcutaneous tissue, they could be seen adjacent to the underlying cartilage, there was no apparent infiltration of the epithelioma among the cartilage cells, thus demonstrating the fact that cartilage cells offer a barrier to the rapid extension of the cancerous process.

An associated infection of the ulcerating mass was clearly indicated by a pronounced leucocytic infiltration.

Over eight months have elapsed since the operation, with no appearance of a recurrence, which usually takes place within two months, and seldom after six months. That the neoplasm was not longer in its growth than thirty days is uncertain. As the man was rather frail in health, and not overly careful in his attention to personal details, his long hair laying over the ulcerating growth, my thought was that the neoplasm, before ulcerating, had probably existed longer unobserved.

As showing the infrequency of cancer of the external ear, Fisher[1] quotes the London Hospital report as stating that of two hundred thousand cases of all kinds admitted each year, but thirty cases of carcinoma of this organ were encountered in a period of ten years' time. At the St. Bartholomew's, seven cases were encountered in two years. The Liverpool Eye and Ear Hospital reports show two cases treated in ten years. The Mayo Clinic had twenty-seven cases from December 31, 1907, to October 31, 1913. In addition, Fisher collected twenty-two additional cases from the literature.

Wolffe,[2] in commenting on the infrequency of cancer of the external ear, quotes Haig in saying that he was able to collect only sixteen cases of tumors of any sort in this situation, while Alexander gathered from the literature fourteen cases; of these, three were fibromas, one a tuberculous granulating mass, one an angioma, three endotheliomata, three epidermoid carcinomata, two hair-matrix carcinomata, and one atheroma.

When carcinoma attacks the external ear, it is usually found to be on the posterior surface. Of seventeen cases collected by Halle, thirteen were situated on the posterior surface of the pinna and four on the anterior. It has been found, however, to be situated on the various parts of the ear, although the lobe of the ear is peculiarly free from assault. Coudray, in Wolffe's Krebskrankheit, reporting a single case only occurring here, and that was of the hair-matrix character (rodent ulcer). R. Haig, in the same work, observed only one case in this situation, and that was a cancer engrafted on the border of a tuberculous mass. Occasionally, a case is encountered that is so far advanced that it is quite impossible to tell the original seat of attack. About three-fourths of the cases of cancer of the external ear are situated primarily on the pinna. When occurring on the posterior surface of the ear, many of the cases have originated in the auriculo-mastoid furrow.

Carcinoma of the external ear occurs almost exclusively in well advanced middle life or in old age, at a time when nutritional changes of a degenerative character are most pronounced. In the Mayo Clinics, quoted above, the average at which the cancer occurred was sixty-two; the youngest forty-six; males greatly predominating; in their series being seventy-four per cent, while those in the literature were eighty-five per cent. Cancer of the pinna does not occur in the young, although Bezold, in Wolffe, reported a case of angiosarcoma of the outer ear in a girl of nine years, the tumor having taken its origin from the concha.

Just as irritative influences stand as etiologic factors in the production of carcinoma in any other situation of the human organism, so here, on the external ear, anything that keeps up a constant irritation, first influences an increased production of epithelial cells, which overgrowth may eventually become lawless in their arrangement.

Inasmuch as carcinomata have frequently arisen in the auriculomastoid furrow, occupied by spectacle bows, these have been thought to exercise a causative influence.

Preceding the development of a carcinoma of the external ear, some of the cases have been the seat of an eczema, especially located on the posterior surface.

In many of the cases, cancer of the external ear was associated with a sailor's skin. As this has unquestionably stood as an etiologic factor in other situations of the exposed integumentary surface, it is only reasonable to conclude that a similar influence is exercised here on the ear by the sun's rays.

The irritative influence of a chronic suppurative discharge and of hardened ear wax is thought to sustain a causative relation, yet this association is not at all constant in cases of carcinoma in this situation.

The squamous type of cell (epidermoid carcinoma), prevails over that of the hair-matrix type (rodent ulcer), in carcinoma of the external ear. The tendency of the latter type[8] is to infiltrate more broadly but less deeply the surrounding tissues in any situation in which it occurs; it is slower in its progress, and is less likely to metastasize than in epidermoid carcinoma, though in either form of the disease this is not inclined to take place except late in the history of the malignancy. In either form, too, the cartilage seems to form a protective barrier against its further progress.

While glandular metastases are not frequent in cancer of this region, recurrences are much higher than elsewhere, occurring at the Mayo Clinic in fifty-eight per cent of the cases. In the literature, reference to this point is more uncertain. Recurrence takes place more quickly and more frequently when the epithelioma is attached to the cartilage than where no such complication exists. Many of these cases have been successfully dealt with by a second operation.

Inefficient removal of the growth is undoubtedly responsible for so many recurrences. The situation of the cancer on the external ear makes one hesitate to do a more mutilating operation than is absolutely necessary. As a result of this, frequently the incision stops short of the real necessity of the exacting conditions present.

The diagnosis of cancer of the external ear is not difficult as a rule. Tuberculosis and granuloma of the canal may simulate carcinoma and require differentiation. Chancres, gumma, and lupus of the external ear may lead to such confusion in diagnosis as to require the removal of a section for histologic examination, to be followed promptly, however, by complete excision of the mass, if its character is of a malignant type; otherwise the cancer may be stimulated to active growth, or permit the dissemination of cancer cells into surrounding tissue or metastasis into neighboring glands; the latter, however, being a remote possibility in recent cases, it not occurring usually in cases under four years' duration.

It is unfortunate that some patients seek relief for cancer of the external ear only after it has been in existence for a long time. In order to make an operation for cancer successful, here as elsewhere, it must be done at a time when the entire growth can be removed completely. As glandular metastases are infrequent in the external ear, the operation offers better opportunities for complete eradication than in most other situations. The great drawback to successful removal is the difficulty of avoiding a mutilating operation on the pinna. The epithelioma is frequently attached to the cartilage, which adds some gravity to the prognosis. To make the operation successful, it is necessary to remove enough of the cartilage to insure the complete excision of the cancer, and to cauterize with heat the entire raw surface, so as to destroy and isolate infiltrating cells. It is not necessary to remove the glands unless they are involved in metastases, and it should not be done as a routine operation.

In the Mayo series of cases, reported by Fisher, four had glandular involvement; two of these were operated on, one recovering, the other died. In the literature, forty per cent showed glandular involvement; five of these cases were operated on, with three deaths. Of the three cases resulting fatally, one had a radical removal of the glands, while the two recoveries had a complete removal of them. Sixty-seven per cent of the Mayo series, without glandular involvement, recovered; in the literature, fifty per cent. There seems to have been no glandular involvement of a malignant type in any of the earlier cases, Fisher reporting it only in cases of at least four years' standing, with seventy-one per cent of the cases showing a primary involvement of the pinna. In his series of cases not cured, all were adhered to the cartilage. Unfavorable conditions to an ultimate cure are, actively growing neoplasms, adhesions to the cartilage and wide or extensive infiltration. As epitheliomata of the external ear are resistent to treatment, an operation should be done early when the greatest prospect of cure is at hand, and the incision made sufficiently generous to make probable an absence of recurrence. Unless it is practicable to remove the neoplasm at a reasonable distance from its attachments, on account of making a too mutilating operation, radium should supplement such operation as one is able to make, first employing the actual cautery.

If an observation of six months fails to reveal a return of a cancer of the external ear, a cure may reasonably be depended upon.

SUMMARY.

1. Cancer of the external ear is of infrequent occurrence. Fisher collected twenty-two cases from the literature occurring in different parts of the external ear and presented twenty-seven cases from the Mayo Clinic.

2. Three-fourths of the cases of cancer of the external ear occur on the pinna.

3. While occurring on different parts of the external ear, carcinoma generally attacks the posterior surface; less frequently, the anterior, occasionally some part of the rim, and in one case, the lobe of the ear.

4. Epitheliomas of the external ear occur in males in seventy-eight and one-half per cent of the cases.

5. Cancer of the external ear, exclusive of the external auditory canal, occurs only in advanced life or in those well into the middle period, when nutritional disturbances of a degenerative character are usually present.

6. Irritative influences, here as elsewhere, are responsible for the initiation of a malignant neoplasm. Eczema, spectacle bows and sailor's skin are all thought to be occasionally responsible for the growth.

7. The epidermoid type of carcinoma prevails over that of the hair-matrix or rodent ulcer form.

8. Insufficient removal is responsible for many of the recurrences; fear of a mutilating operation is usually responsible for the insufficient removal.

9. After the removal of the growth, the surface of the wound should be gone over with the actual cautery.

10. If the surface is broadly infiltrated, the operation should be followed up with the use of radium.

11. Glandular involvement is not usual, and it is a late complication when it does occur. Out of twenty-seven cases in the Mayo series, it occurred four times; in the literature, forty per cent of the cases showed this involvement. In the former

cases, it did not occur in any of them which had been in existence less than four years.

12. In cases of supposed eczema of the external ear occurring in advanced life, one should be thoroughly alert for cancer; if found, operate early enough to be a real service to the patient.

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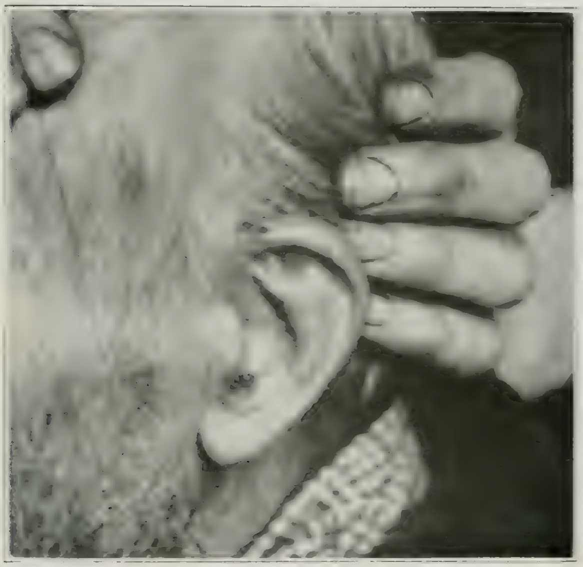

FIGURE 1.

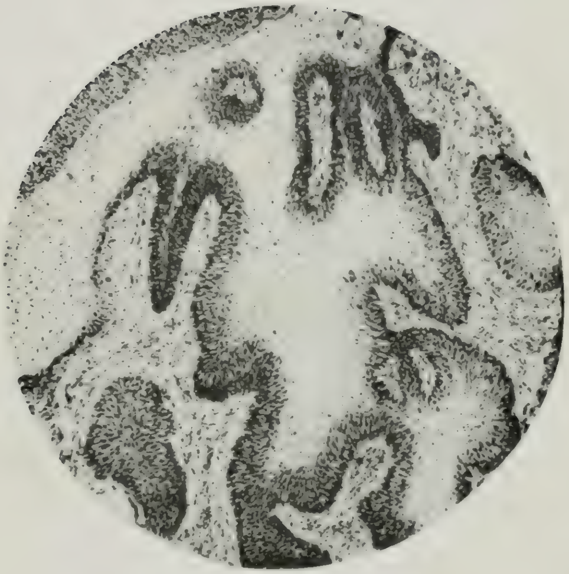

FIGURE 2.

LIX.

ABSCESS OF THE LUNG FOLLOWING OPERATION ON THE TONSILS AND UPPER AIR TRACT.*

By Charles W. Richardson, M. D.,

Washington, D. C.

It seems unnecessary for me in this paper merely to call attention of the members of this society to the papers that I have written bearing upon this subject before other organizations, as I feel that they have been quite generally read. On March 20, 1912, I believe I first called attention to this condition as a complication of tonsillectomy. Since the publication of my paper on "Abscess of the Lung, following Operations on the Upper Air Tract," in the *Laryngoscope,* July, 1916, I have received a number of communications from observers throughout the country, stating that they had met with this untoward experience in their tonsillectomy work. Having been established that this serious complication is not now an unusual sequela of operative work, it therefore, becomes incumbent upon us to ascertain the manner of its occurrence, and to consider wherein we may adopt alterations in our methods or technic by which it may be prevented.

To all observers it must be patent that this complication has largely come under our observation since we have adopted the more radical operation of tonsillectomy in dealing with enucleation of tonsils. It must also be patent to every one that while we do a great many more radical operations upon the sinus than formerly, in which there is free pus, granulation tissue and infective débris, which must in certain amounts gain ingress into the air tract, yet we do not find that lung abscess in an increasing factor in these operations.

What is the probable etiologic factor that enters into the causation of this complication?

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*Read before the American Laryngological, Rhinological and Otological Association, May, 1917.

Under the various causes assigned as the factors in producing pulmonary abscess are:

First. General anesthesia.

This is only a remote factor, in that the anesthetic irritates the pulmonary mucous membrane and renders it more susceptible to the invasion of pathogenic germs that may have gained ingress, and, through rendering the patient unconscious, it lowers the expulsive reflex, thus lessening the great natural defense to the protection of the air tract.

Second. The want of thorough examination and preparation of the patient.

This factor should never enter into the causation. I was astonished in my reading of the report of the New York Academy of Medicine, Section on Laryngology and Rhinology, meeting of January 26, 1916, of the discussion on this point. There is and can be no excuse for the want of examination and preparation of patients about to enter into operative procedure upon the upper air tract. My patients are always examined physically. The orders for the preparation are always written out and given to each patient with injunction to follow explicitly. I have never had a patient to vomit on the table. The bowels are always freely opened, and they have no food for at least eighteen hours before the operation. The urine is always examined.

Therefore, after a most thorough consideration of this subject, it seems to me that the method of invasion is either indirect, through the lymphatic or venous system from the wound surface, or direct, through the inspiration of pus or bacterially laden caseous material into the pulmonary tract. By whatever method we perform this operation there is more or less squeezing of the tonsils so that contained pus or masses of cheesy débris are forced out from the substance of the tonsils or from the diseased crypts. Even while making the incision to liberate the folds of the tonsils, we have on numerous occasions observed pus, more particularly cheesy concretions, floating out on the surface of the tonsils. This occurs almost as frequently with children as with adults. By whatever method we perform the operation, we necessarily open up numerous lymphatic vessels and moderate size veins, and leave a deep wound surface which is bathed in the abundant flora of the faucial cavity.

If these are the two important factors which I believe they are, we should make all efforts to minimize their effect by lessening the amount of trauma as far as our operative skill will permit, and through adopting all methods to prevent the ingress of pus or cheesy material from the tonsils into the pulmonary tract.

I had hoped through the various improvements of my own technic to exclude this complication from my operative risks; but my work during the past year has shown me that I have not succeeded. In spite of my depression of the patient's head, through the raising of the foot of the operating table thirty-five inches, and the use of the suction ether apparatus of the Beck-Mueller design which has always worked most satisfactorily—I have had one abscess formation. One interesting feature to me in this connection is the fact that I have never seen an abscess of the lung following an operation upon the upper air tract in a child under fourteen years of age. In the Children's Hospital, in Washington, in which the entrance age is under twelve, and wherein I do for six months all the open ward, and throughout the year many of my private children patient's operations for tonsillectomy, and where we do not have the advantage of the suction ether apparatus, I have never had a pulmonary abscess.

All my adult operations are done at the Episcopal Eye, Ear and Throat Hospital, as well as some of the older children, under the greater advantages of position and suction. The number of my operations done in each institution is about equal. Is there some anatomic relation in the fauces, or is the resistance power in the lungs in a child greater, which renders it less susceptible to this type of infection than the adult?

From the fact that children seem totally unsusceptible to this type of infection, from the fact that infection occurs as a result of submucous resection, which is an absolutely clean operation, and that it does not transpire with any degree of frequency after opening up such pus bearing fields as the nasal sinuses, I am still inclined to believe that this complication occurs in the fair proportion of cases by infection through the lymphatic or venous channels. Another strong factor that points to this manner of infection is the fact that most of the abscesses that I have seen radiographed have apparently developed between the lobes of the lung—probably from the interlobular lymph spaces.

With these facts before us, should we not seek to modify our operative procedure upon the adult? Is the patient under general anesthesia more susceptible to this form of infection than under local anesthesia? Is the occurrence of faucial and deep cervical infection, after local anesthesia, as frequent and as dangerous to the patient as the occurrence of occasional lung abscess in the general anesthetized adult? The above question can only be answered after a thorough survey of the situation and upon better knowledge of the advantages offered through the two methods that we now possess.

SYMPTOMS OF LUNG ABSCESS FOLLOWING OPERATION UPON THE UPPER AIR TRACT.

The earliest and most characteristic symptoms is that the patient does not make a favorable convalescence after the operation. The case progresses favorably with regard to local manifestations, but the patient seems languid, somewhat dispirited, and inclined to remain abed. Usually from the fourth to the tenth day positive evidence of pulmonary invasion makes itself manifest. We have irregular septic temperature, sharp pain in the lung, and violent paroxysmal coughing. Occurring at the same time, or shortly thereafter, we have the pathognomonic evidence of pulmonary abscess in the expectoration of a free pus which has a foul odor. This foul odor is intensely distressing to the patient and is noticed by them, not only with the expectoration of the pus, but also during coughing. In some cases the odor is noted by the patient some hours before the expectoration of pus. In the early stages, and frequently throughout the case, the foul odor is only noted by the patient. The physical signs at this stage are very meager, depending upon the size and location of the area involved. These signs are dullness over the area affected, diminished breath sounds, and crepitant or subcrepitant rales. There is no doubt that some cases at this stage are supposed to be ether pneumonia. With the development of the abscess we have characteristic symptoms manifested—viz., the expectoration of pus, pain, greater or less actual hemoptysis, and foul odor. In the several cases which I have had under my observation, although the patient complained bitterly of the odor, the odor of the breath was never noticed as disagreeable, although the expectoration from the same patient had a distinct, unpleasant odor. The cough persists, nausea is frequently present, the fever becomes septic, the fingers club and the patient gradually loses flesh. The physical signs at this stage are those of lung abscess. Radiograms taken at any time will show the graphic evidences of the abscess.

Treatment.—The management of pulmonary abscess may be considered under three classifications—viz., medical, or expectant; surgical, or compression of the lung by artificial pneumothorax.

In a small percentage of cases when the abscess is located in the upper lobe of the lung and the drainage is into a large bronchus, and where the case is apparently following a favorable course, the medical expectant treatment may be followed. It is well to have in mind the fact that the percentage of mortality is very great by this line of treatment, approximately sixty per cent, and therefore it must not be pursued for too long a period.

The surgical method, consisting in rib resection, fixation of lung and drainage of pus, gives a much better result than medical treatment, the mortality being approximately thirty per cent. The more radical surgical procedure of excision of portions of lung containing the abscess cavity, indeed of one or more lobes, has been attended with excellent results The surgical treatment has offered the best results heretofore, although with a fairly high mortality and frequently a prolonged drainage before the case is brought to resolution.

Artificial Pneumothorax.—By this method we have apparently the ideal, rational and most acceptable method of treating acute pulmonary abscess. As is well known, the method has been for a long time used with more or less marked success in the treatment of chronic pulmonary abscess. Dr. W. D. Tewksbury, superintendent of the Washington Tuberculosis

Hospital, I believe, was the first to suggest its use in acute pulmonary abscess, although he gives credit to Dr. A. E. Greer, to priority in the publication of a single case of interlobar empyema following pnuemonia, treated by compression of the lung with nitrogen. Dr. Tewksbury in his production of artificial pneumothorax employs air instead of nitrogen.

Dr. Tewksbury in a paper read before the Medical Society of the District of Columbia, entitled, "Acute Pulmonary Abscess Treated with Artificial Pneumothorax," November 8, 1916, gives the results of treatment in his first two cases. He has kindly furnished me with the data and results obtained by him in treating acute pulmonary abscess since the publication of his above mentioned paper. In the past ten months he has seen ten cases of acute pulmonary abscess, nine of which followed operative treatment upon the upper air tract, by eight different operators, in eight of which he employed artificial pneumothorax. In two cases expectant treatment was attended with resolution. It is interesting to note that these cases were operated upon by eight different operators, each employing a slightly different technic, so that the result cannot be assigned to a universally employed technic. These results also demonstrate quite a large proportion of lung abscess for a community as small as Washington, and bear out the statement that I made in my paper read before the American Climatological and Clinical Society, May 9, 1916, "but while these remote lesions are rare, I am convinced that they are not so infrequent as one would judge from reports hitherto made from the domain of laryngorhinology."

Dr. Tewksbury's conclusion is as follows:

Of the eight cases treated by artificial pneumothorax, six were acute lung abscess of a month or less duration. Of this number four recovered, one is recovering (at this writing well), and one died. Two were chronic lung abscesses of over a year's duration. Both improved, but were not cured.

It is apparent that in order to get cures in these cases it is necessary to use pneumothorax early while the abscess is soft and compressible.

LX.

REPORT OF INTERESTING NASAL CASES DUE TO SYPHILIS. REMARKS ON OBSCURE SYPHILITIC NASAL SYMPTOMS.*

By Dunbar Roy, M. D.,

Atlanta.

Obscure syphilitic manifestations in the nasal cavities are much more frequent than many of us suppose. Since the introduction of the Wassermann test the writer believes that we have made ourselves too dependent upon it as the final test in diagnosis, to the exclusion of clinical symptoms which are so important in the management of pathologic conditions. The following case is presented in detail as affording many points of interest to the neurologist, the internist, as well as to the rhinolaryngologist.

Mr. E. E., aged fifty-one years, cotton merchant. Patient had always been strong and healthy, never having had a severe spell of illness during his life. Is temperate in both drinking and smoking—in fact, seldom takes a drink. Has lived in Atlanta most of his life. His father and mother are both living, and both in excellent health. In August, 1902, he moved to Shreveport, Louisiana, to represent a northern firm as their cotton buyer. In that city he seemed to enjoy good health, remaining at his usual weight of one hundred fifty pounds. Patient was not over four feet nine inches in height. He spent each summer in Atlanta, returning to Shreveport in the fall. During the winter of 1903-04 he did not feel in his accustomed good health, and along in the spring of 1904 he began to have neuralgia and drawing sensation in the muscles in the back of his neck. In May, 1904, he returned to Atlanta again to live, having made new connections in the cotton business. On arriving in Atlanta he consulted his physician on account of this severe neuralgic pain in the back of his neck. His appetite was not good, and he was already beginning to lose flesh. Being a neighbor and a warm personal friend of mine, I saw him quite frequently, but in an entirely nonprofessional manner. In this way I noted his gradual decline. His physician attributed the trouble to muscular neuralgia, as a result of malarial intoxication. He was now treated steadily, but without improvement, while the pain gradually extended down into his shoulders and arms. These pains grew to be excruciating and would come upon him in paroxysms. His digestion and appetite were poor, and his nightly sleep very much disturbed. Not improving, he called in another physician (which is characteristic of a great many patients, and we can hardly blame them), who took charge of his case. He also attributed the cause to malaria, and immediately began to dose him with various medicines, changing the same from time to time. He was put upon a strict diet, and his liver was kept very active. Soon a derangement of the function of this organ was diagnosed as playing a prominent rôle in producing the various physical ailments. The patient was blessed with an excellent nurse in the shape of his wife, who rubbed and massaged him with various liniments and looked after his every physical and mental comfort.

The pain, however, did not subside, but was extending still further down the patient's back, and even into his legs. At no time were there any signs of paralysis, and the patient was able once a day to go to his office and remain for a few hours.

In giving the history the patient told me that his physician made a thorough examination of his secretions, urine, etc., and found nothing abnormal. The pains in the shoulders, especially the left, and in the back were sometimes accompanied by such spasms of the muscles as almost to draw the head to the shoulder. In September, five months later, as he was not improving, but seemingly growing worse, becoming weaker each day, losing flesh and the pains being so severe as to render sleep impossible except for a few hours at night by means of codein, he consulted a renowned osteopath, who told him it was due to the nerves from the spinal column. He gave him three treatments a week and put him upon a still more restricted diet. This treatment he continued for a month, having discarded all medicines except certain mineral waters suggested by friends. At the end of this time he was no better in any respect, the pains not having been relieved, they being now in the legs as well as in the arms, and accompanied by the most violent spasms. At this point the patient began to be hoarse, but without cough or expectoration.

During a social visit one evening he remarked that he had taken a severe cold, and if he did not get better was coming to me professionally. In the meantime he continued to have thorough bodily massage, but was dependent upon medicine to bring sleep. It was frequently remarked among his friends that Mr. E. looked like a doomed man, and they believed that he would live but a few weeks. On my return to the city after the Christmas holidays, his wife telephoned that Mr. E. wished to consult me in reference to the severe cold in his head and throat. Being a personal friend and a neighbor, I was really sorry that the patient had come to me for professional advice and treatment, for with his previous history in regard to his medical treatment (of which I was cognizant) I felt that I could do but little for him now that he seemed in the last stages of some obscure nervous disease.

When he came into my consultation room he was so weak, feeble and emaciated that I felt as if he would never be able to return to his home. His weight then was ninety-five pounds, having been reduced from one hundred and fifty. He had no signs of paralysis, his reflexes were normal, and his only complaint being the intense pain and spasms in the legs and arms, accompanied with the gradually increased weakness.

Two weeks previous, according to statement, he contracted, in addition to the hoarseness, a cold in the head, and this had gradually kept up until at present it was quite disagreeable, he having hardly sufficient strength to blow the tough secretion from his nasal cavities. His throat, also the larynx, worried him considerably by being so tight as to interfere with his breathing, especially at night.

Examination of Nasal Cavities.—At the mucocutaneous surface in both nostrils the parts were thickened and inflamed from irritation produced by the nasal discharge. The right cavity seemed pretty well filled with mucopurulent secretion. After shrinking the tissues with cocain and adrenalin, and by assistance from both patient and myself, the cavity was fairly well cleaned. Far back on both septum and middle turbinal there was a dirty yellowish membrane covering the parts which extended also into the nasopharynx. A slight manifestation of the same condition was found on the left side, but not quite so marked. The parts bled when some of the membrane was detached. By means of cotton on the end of an applicator I detected roughened and exposed bone high up on the septum at its posterior free border, and also the same condition at the posterior end of the middle turbinal on the same side—i. e., the right. None was detected on the left. The pharynx showed nothing abnormal, but there was a large amount of tough yellowish mucopurulent secretion coming down from the nasopharynx and clinging to the postpharyngeal walls. It was impossible to inspect the nasopharynx on account of the relaxed condition of the palate and the gagging produced in my efforts to use a palate retractor.

On inspecting the larynx I found a beefy-red swollen condition of the whole laryngeal mucous membrane, the cords being of the same general color. The membrane there and further down in the trachea seemed to be producing a tough, membranous secretion which it was impossible for the patient to expel. It gave the sound of a "croupy cough." He had no pain either in his throat or nose, but complained some of a pain just at the base of the nose over both frontal sinuses. Transillumination of these sinuses showed no shadow at any point.

The objective picture here presented was to me quite significant of a specific condition, but close inquiry elicited not one ray of such a history. I am sure the patient would glady have told me if such had been the case, knowing that the treatment then would be much more effective. I knew him intimately, and there were few men who lived a life of more sobriety. Even before his marriage he was never given to the slightest dissipation. The only other condition which such symptoms were likely to represent was tuberculosis, and this to me almost seemed the correct diagnosis. I had the secretion from the nose and throat examined, but this was negative for tubercle bacilli. The loss of weight, hoarseness, slight cough, were symptoms quite significant, especially after the negative specific history. However, I knew that histories were often uncertain, and as an experiment in conjunction with the local treatment of cleansing and nitrate of silver applications, I put the patient on the following treatment: On account of the poor appetite and atony of the gastric function, he was given ten drops of equal parts of tincture of nux vomica and compound tincture of gentian in a little water and corn whisky before meals. After meals he was started on five drops of a saturated solution iodid potash in one-half glass water, and this was increased one drop every other day. In the morning and afternoon he was given an expectorant mixture of carbonate of ammonia, fluid extract of squills, etc., this being used to soften and aid the expulsion of the tracheal and laryngeal secretion. He was not limited to his diet, but told to eat as much nutritious food as possible. He was also to use a douche of warm saline solution in the nasal cavities three or four times daily.

The next day being very cold, patient did not report, but his wife telephoned me that a swelling had developed just at the root of his nose, accompanied by some pain on pressure. In the evening I called and found the patient sitting up in a big chair with a swelling, puffy and globular looking, about the size of a walnut, just at the root of the nose. It had developed slowly during the day. I advised hot fomentations every hour, a good saline internally, and the other treatment to be continued as prescribed. The next day when seen, the edema or swelling had extended to the lids of both eyes until they could not be opened and even down on the cheek and nose. The original point of the swelling was beginning to subside. The following day, Dr. Block, a neurologist, was called by me in consultation. Dr. Block made a very thorough physical examination of his heart and lungs, muscles, nervous reflexes, and urinary secretions. This latter was found to contain a trace of albumin but no casts.

Dr. Block was unable to suggest anything further except to give the patient a mixture containing the bromids, to be taken at night for his extreme nervousness. Edema remained limited to the face, and this gradually disappeared entirely in one week.

From this time on the patient began to improve and the internal remedies were never changed, the iodid being increased to fifty drops three times daily. I saw him at my office every other day, cleansed thoroughly the nasal cavity, touched the necrosed condition in the nose and nasopharynx with a strong solution of nitrate of silver, followed finally with a covering of aristol powder. Inhalations of terebene in benzoinated alboiene were given for the larynx and trachea. One morning about three weeks after the beginning of the treatment, the patient blew from his nose a mass of secretion and a small piece of bone which proved to be a part of the vomer.

The patient was treated at gradually varying intervals until August 1, 1905—i. e., seven months—when he was dismissed, apparently cured except that he was still hoarse from the hypertrophied condition of the laryngeal mucous membrane. I tried astringents and sprays, but this seemed to do but very little good. The medicine was never changed during the whole course of treatment, the remedies being gradually left off with the improvement of the patient's condition.

December 28, 1905, one year after I first saw him professionally, he weighed one hundred and fifty pounds, and with the exception of slight hoarseness he was as well and as strong as at any time during his life. There was no discharge in either the nasal cavities or the nasopharynx. Up to the present time there has been no further trouble. He has been in the best of health now for fourteen years.

Remarks.—This case presents many interesting as well as obscure features. In the beginning of the patient's trouble there were some symptoms of a malarial neuritis; in fact, all the physicians consulted during the first months of his illness attributed the cause of his indisposition to malaria. The very first symptom noted by the patient was a general feeling of malaise and loss of appetite. This occurred during the last months of his residence in Shreveport. In May, 1904, he returned to Atlanta weakened in health and suffering with drawing pains in the muscles at the back of his neck. There was never any rise in the bodily temperature throughout the course of the disease, nor was there at any time signs of paresis in any portion of the body. It is just possible that a slow grade multiple neuritis might produce some of the symptoms in this case, but my own observation and that of other observers would not lead us to such a conclusion. For indeed at no time during the illness was there signs of paresis, but most prominent were the signs of spasms and intense neuralgia of the sensory nerves.

The diagnosis of malarial intoxication was certainly a most natural one, and it is still a question in my mind whether the beginning trouble could not have been attributed to this cause.

The brilliant result obtained by the internal administration of iodid of potash almost compels one to acknowledge the specific basis of the whole constitutional derangement, and yet with the exception of this one fact, the good results obtained by the internal administration of the iodids, there was absolutely no history or symptoms to warrant us in drawing such a conclusion. I examined him thoroughly at the time, and even since then there has not been the slightest signs of glandular enlargement of any portion of the body. No falling out of the hair, no symptoms of sore throat, no obscure pains at night, absolutely nothing to warrant us in suspecting syphilis as the cause. So foreign to my mind was syphilis as the cause of the nasopharyngeal ulceration at the time of these very active symptoms, that I concluded the ulceration was without doubt tubercular, on account of the great loss in weight and the seeming involvement of the bronchial tubes. However, Dr. Smith, an excellent pathologist, made a thorough examination of the secretion with absolutely negative results. His mother had been treated for years for a superficial skin cancer on the forehead and this is still under the constant observation of a dermatologist.

Another symptom which was misleading to me was the existence of an odor from the nose in nowise characteristic of that experienced in the cases of late tertiary syphilis, where the odor is exceedingly offensive, and especially where there are areas of necrosed bone. While many things point to the correctness of our diagnosis, we must remember that the administration of the iodid of potash often proves of benefit and even curative in cases other than syphilis as, for instance, an alterative in various conditions where we can give no better name than "blood dyscrasia", this remedy is often of marked value. As an adjunct to the relief of various inflammatory exudates in the body, I know of no one remedy that is of greater value.

Consequently, because the case responded to the iodid of potash, and seemingly was brought to a successful issue through such administration, is no self-evident proof that we were correct in our diagnosis.

The question could very naturally arise, was malarial intoxication the primal cause of this physical derangement, and did its presence in the system so impoverish the blood and thereby the nerve tone as to produce the train of symptoms which occurred throughout the course of the disease? In my own mind there is such a possibility, and yet in studying the literature of any similar case reported, I can find nothing which would lead me to such an affirmative answer. After all that can be said, the history of this case and the results obtained by the treatment certainly bear out the probable diagnosis of its specific character.

Another case evidently specific in character, occurring in my practice several years ago, had many obscure points in its diagnostic history and yet the final results must lead us to the same conclusion as was obtained in the first case.

The second case is that of a Mrs. M., aged fifty-five years, widow. This patient was a woman of most excellent standing. She was the mother of four children, all of whom were unusually robust in their physical growth. The family history precluded any idea of specific taint. The father and husband died several years ago of typhoid fever. The patient had always enjoyed the best of health and had never suffered from any of the feminine complaints.

Present History.—In April, 1903, patient consulted me on account of cold in the head which had been present for two weeks and which did not improve after the use of all the ordinary remedies. Her chief complaint was a stuffiness and inability to breathe comfortably through the nasal cavities. There had been some symptoms of tenderness about the external part of the nose whenever the handkerchief was used. With this exception her general health was perfect and there had been no signs of constitutional derangement.

Examination showed some swelling of the whole outside of the nose, giving her the appearance of the socalled "frog-face." Nasal respiration was difficult, producing the whistling sounds so characteristic of an intranasal swelling. On examination with reflected light the anterior part of the septum was thickened and edematous, resembling very much the appearances seen in an abscess of the septum. This could not be reduced to any appreciable extent by the use of cocain and adrenalin. The outer walls, including the turbinates, were only slightly swollen. There was no history of traumatism. This was before the time of the Wassermann test, and hence this could not be used. The question of malignancy could very readily be suggested. There was no bleeding when the septum was touched, and only the thick leathery sensation was imparted to the nasal applicator. There was very little space left between the septum and the outer walls. The pharynx and nasopharynx were absolutely normal in appearance. No glandular enlargement could be elicited. However, the latter seemed to me the most probable diagnosis from all the clinical features of the case.

Treatment and Results.—The patient was placed upon rapidly increasing doses of the iodid of potassium. The fact that this medicine was tolerated with the greatest ease indicated somewhat that the condition would respond to this line of treatment. Almost immediately the parts responded, and in a few days the patient began to loose the stuffy feeling in the nose and respiration became much easier. No other treatment was used with the exception of an oily spray, which was only for comfort.

The treatment was continued for three months at the end of which time the nasal cavities were perfectly normal in appearance, and up to this time, thirteen years, there has been absolutely no return of this or any other condition resembling a complication of specific dyscrasia.

Remarks.—Of the certainity of diagnosis there of course may be some difference of opinion, but the results obtained by the use of the iodid of potassium would rather indicate the specific nature of the affection. As was stated at the beginning of this paper, the writer believes that there are many obscure lesions in the nasal cavities which have their dependence upon syphilis. It is not always necessary to look for ulcerations and even destructive processes in order to have a picture of nasal syphilis. Manifestations of specific lesions in the nose resemble many other affections, and the fact that we sometimes get such poor results in intranasal operations may have its real cause in a failure to recognize a constitutional systemic dyscrasia which in one word is syphilis. The rhinologist who waits for typical textbook pictures of lesions, or is entirely dependent upon clinical tests, will fail in many instances to bring about a successful result. The obscure ramifications of the syphilitic virus are manifold in character, and we can never be too diligent in instituing antispecific treatment, if no other cause for the condition can be found.

Congestion and edema of the nasal mucous membrane, as is sometimes found in the early stages of syphilis, very frequently resembles those cases of vasomotor rhinitis in all its manifold symptoms.

A typical case of this kind occurred in my practice three years ago. Mrs. E. E. L., aged forty-four years, consulted me in February, 1915, with what she denominated a severe cold in the head. Patient was confined to her bed, complaining chiefly of the inability to breathe through the nose, frontal headache, sneezing, all the symptoms of acute rhinitis. Patient said she had been having periodic attacks of this kind for the last year.

Examination showed congested and edematous turbinates with a serious discharge. Under local applications of cocain and adrenalin the membrane was with difficulty contracted. The writer has always found such a condition characteristic of vasomotor rhinitis and syphilitic congestion. Ordinary rhinitis will show contraction immediately under cocain applications, thus differing from the two former conditions. The usual treatment was instituted, but with very slow and imperfect results. The patient was under the writer's care for several weeks, and it was only by continuous treatment that she was made even partially comfortable. The greatest complaint was the stenosed condition of the nasal cavities at night. This was relieved during the day, especially when the patient was out in the open. This symptom being characteristic of vasomotor rhinitis, it seemed as if this latter condition was the proper diagnosis. However, the patient's progress was exceedingly slow, and there were repeated exacerbations of all the nasal symptoms. An examination made two months after the first observation of the patient revealed a small superficial ulcer on the septum opposite the middle turbinate on the left side. Syphilis had not been suspected on account of the excellent social standing of the patient. A Wassermann was immediately made which was returned plus three positive. This patient was immediately placed upon rapidly increasing doses of the iodid of potassium, with a corresponding improvement of her condition. The ulcer healed immediately, and complete relief from all the symptoms of nasal stenosis rapidly disappeared, together with the accompanying vasomotor disturbances.

It is now three years since this attack and the patient has had no further trouble.

In conclusion the writer cannot urge too strongly the importance of the syphilitic virus as the fundamental cause of many obscure rhinologic conditions which resist the ordinary treatment. Nor should we be governed entirely by such tests as the Wassermann in directing us to the proper line of treatment. During the last twenty years the writer, as the head of a large free clinic, where the majority of patients are negroes, and where he has seen syphilitic lesions of every known variety, has adhered entirely to the old treatment of iodid of potassium and mercury, and with few exceptions the results have been entirely satisfactory if this treatment is given from year to year.

Grand Opera House.

LXI.

REPORT OF A CASE OF MENINGITIS WITH SOME UNUSUAL FEATURES.*

By Chas. E. Perkins, M. D.,

New York.

The following case is reported in the hope that on account of some uncommon features it may be of interest to the society.

Wm. G., aged nineteen years, was admitted to Dr. Dench's service at the New York Eye and Ear Infirmary, under the writer's care, on August 13, 1917, with the history of having had a discharge from the left ear since an attack of measles nine years previously. The discharge possessed odor, varied in amount, but was more or less continuous.

On admission, the patient's temperature was 103° and he appeared very sick. There was nearly complete absence of the drum membrane, no apparent sagging of the canal wall, the otoscopic examination indicating nothing as to the severity of the process. The discharge was rather scanty and a smear showed mixed infection.

The mastoid did not seem tender, and there was no postauricular swelling or edema. No headache, stiffness of the neck, or Kernig. He answered questions readily and rationally and his mental condition seemed good. Hearing left ear, whisper, three feet; right ear, whisper, ten feet. Caloric reaction positive on both sides. No nystagmus, vertigo, or overpointing. Blood showed leucocytosis of 28,000; polymorphonuclears, percentage of eighty-six. Culture proved negative. Spinal fluid flowed under about normal pressure, but was so mixed with blood that the cell count was of very slight if any value. It contained 658 leucocytes to the cubic millimeter. Dr. Dixon thought that the white cells were in greater number than could be accounted for by blood admixture. The culture of this fluid was negative. The globulins were present. Fehling's positive—i. e., sugar was present. Wassermann negative. Spinal fluid on two subsequent occasions contained too much blood for a count; contained globulins, Fehling's plus. Negative cultures. Dr. Dixon's report on the X-ray was: "The right is a very large clear pneumatic bone; the left a sclerotic looking bone with an appearance of epidural abscess in the middle fossa."

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*Read before the New York Academy of Medicine, Section on Otology, December 14, 1917.

A thorough examination of the patient showed absence of any disease to account for his condition outside of the mastoid, so operation was determined upon. On the following day he developed a postauricular swelling, noticed just before operation. His temperature on this second day had mounted from 102.4 in the morning to 105 at noon.

Operation.—Upon making the initial posterior incision there was a hissing sound caused by the escape of foul smelling gas and pus. A regular radical operation was done. The sinus plate was removed, showing a perisinus abscess with collapsed sinus. Large exposure of dura in the middle fossa revealed nothing abnormal except a collection of cheesy-like material in the region of the tegmen tympani.

Smear culture of the mastoid pus gave a large number of germs, fusiform bacilli predominating and a few streptococci.

Morning following operation temperature dropped to 101° and patient seemed better; 12 m., 104.6°; 4 p. m., 100°; 6 p. m., 105.8°. Blood contained 30,000 leucocytes; 81.1 per cent, polymorphonuclears. Culture proved to be negative. At this time, on account of the marked remittance of the temperature and the absence of symptoms of meningitis, as well as the condition of the sinus revealed at first operation, jugular resection was done. The outer wall of the sinus was necrotic, and the sinus contained a dark, foul smelling fluid; bled freely from both ends.

The following morning, the 15th, the temperature was down to 100.6°, rose again at noon to 104°, evening still higher, the jugular resection having apparently no effect either way. He also developed a jerking of the arm and leg on the opposite side, and it was evident that their power was much impaired. He became drowsy. On the 16th he was still worse, and I believed all had been done for him that could be. Nevertheless, on the 17th I did a decompression and, as shown at autopsy, stopped just short of evacuating the large accumulation of pus over the motor area.

His temperature during the last two days of life varied from 104 to 107 degrees, just before death on the afternoon of the 17th.

Dr. Dixon's notes of the autopsy follow:

W. G., nineteen years, No. 13615.—Service of Dr. Dench; died August 17, 1917.

Autopsy made 7:30 p. m., August 17, 1917: Superficial examination showed site of mastoid operation, also operation for resection of the internal jugular vein, both on the left side. When the skull cap was removed, the dura was found to be considerably thickened and adherent about the edges of the mastoid wound. When the dura was removed a great quantity of exceedingly foul pus was found over the left motor area. This shaded off below, anteriorly and posteriorly. There was a little purulent meningitis also on the right side. When the brain was removed a considerable amount of pus was found at the base, most of which seemed to have drained to this point; but there was also an exudate about the pons. No thromboses were found in any of the sinuses. The sphenoidal cells were opened and found normal. As the dura was stripped from the remainder of the petrous portion of the left temporal bone a small amount of cheesy material was found in the region of the superior semicircular canal. There was no evidence of direct extension, though it was assumed that the path was through the dura at the point where the operator found a cheesy mass at the first operation—namely, epidural and just above the tegmen antri.

An exudate was noticed over the inner part of both cerebellar lobes, as well as inner aspect of the left occipital lobe. There was considerable hemorrhage in the arachnoid space over the left frontal lobe, extending back over the fissure of Silvius to the anterior part of the temporosphenoidal lobe. There was also a similar hemorrhage over the outer aspect of the left cerebellar lobe.

No abscess could be found anywhere except a very small superficial one, about the size of a pea, on the under surface of the left temporosphenoidal lobe, which would about correspond to the tegmen antri.

Three germs were isolated from the meningeal pus—staphylococcus aureus, a rather large bacillus of the proteus group, a small Gram negative unidentified bacillus and an occasional colony of streptococcus pyogenes.

Conclusion.—This patient evidently had a purulent meningitis for some time before admission, yet, except for his temperature, which was anything but characteristic, gave no symptoms which we are accustomed to expect in meningitis. Moreover, the cytologic findings did not give us any help. It is true that the presence of blood in the fluid taken at three lumbar punctures made the leucocyte count of practically no value, although Dr. Dixon was inclined to believe that the leucocytosis was out of proportion to the blood admixture, even if the blood did contain 28,000 white cells to the cubic millimeter. The blood also would have caused a positive globulin test, so whether the globulins found present were all from the blood, or partly from the meningitis, no one could have stated. The Fehling's test also failed us, as did the cultures, which were negative in the case of the three spinal fluids.

Another feature was that a process found to be so general should have caused so few symptoms which, except for the prostration and fever, were practically nil until about thirty-six hours before death, when the large purulent accumulation over the motor area gave rise to spasm followed by paresis of the arm and leg.

The escape of gas upon making the initial incision in the radical operation is, to say the least, a rare occurrence, and caused me to suspect that I was dealing with gas bacillus infection, but this I believe was not borne out by the results of culture of the meningeal pus.

Finally, with a sinus in a necrotic condition, filled with a broken down, foul smelling clot, the blood cultures all proved to be negative, and jugular resection seemed to have no influence on the process. One would have expected the meningitis to arise by infection from the sinus, but at autopsy the inner wall was found to be in fairly good condition, although at operation the outer sinus wall was necrotic. Moreover, the meningeal infection appeared at postmortem examination to have extended from the small extradural accumulation of cheesy pus, which was indicated in the X-ray plate and found at operation to exist immediately above the tegmen tympani and antri.

616 Madison Avenue.

LXII.

THE INTRANASAL OPERATION FOR THE RELIEF OF CHRONIC DACRYOCYSTITIS WITH SOME DEDUCTIONS FROM TWENTY CASES.

By Carl F. Bookwalter, M. D.,

No attempt will be made in this brief paper to describe the operation in detail, since it is not a new operation and has been described very well by J. M. West and others. General conclusions cannot be drawn from twenty cases, but some important points may be brought out.

It often has been said that this method gives no better results than those methods having for their object the removal or obliteration of the lachrymal sac. Several ophthalmologists of experience have told me that they invariably get good results by removing the sac. Others of equal skill and experience have said that suppuration is not always stopped by the sac operation and that in many cases tearing is a disagreeable feature. Of course, it might be urged that failure to stop the suppuration is due to poor operating, but tearing frequently follows removal of the sac, even in cases where suppuration has been arrested and the results, except for tearing, are all that could be desired.

Drainage of the lachrymal sac into the nose is not a new idea. Various instruments have been passed through the canaliculi into the sac and then into the nose. Only small openings could be made in this way and such openings soon close. The operation that I have used is essentially that devised by J. M. West. West's first cases were done in the Johns Hopkins Hospital in 1907-1908, but most of his work has been done in Berlin. The object of the intranasal operation is to establish a permanent opening from the nose into the lacrimal sac and nasal duct. Drainage thus obtained relieves the suppuration, and a way is provided for tears to pass into the nose. The opening is made just in front of the anterior end of the middle turbinate and above the inferior turbinate. The upper end of the opening is at the level of or a little above the attachment of the anterior end of the middle turbinate, the lower end is well down to the inferior turbinate. The width and direction of the opening correspond to the width and direction of the sac and nasal duct.

In exposing the bone to be removed in the operation, it is not necessary to remove any turbinate tissue or to destroy any of the nasal mucous membrane except over the intended opening. After the bone is exposed, enough of the nasal process of the superior maxillary and of the lachrymal bone should be removed to expose the full width of the sac and duct. The opening in the bone should extend nearly to the top of the lacrimal sac and almost down to the inferior turbinate. As soon as any of the sac is exposed, a probe should be passed through the canaliculus into the sac. The nasal side of the sac can then be moved by the probe and a good guide for the operator is established. When the sac is well exposed, a small hook serves best to grasp it and the exposed nasal side of the sac and duct can be cut away with a small knife, leaving the lateral side in place. Small punch forceps set at various angles are very useful to trim off any tags or rough edges of the sac. The openings in the nasal mucous membrane, bone and sac should be of the same size when the operation is complete. The nose is lightly packed for twenty-four hours. The operation is rather difficult to do, and to get a good result the operator must be very careful and painstaking. A little bleeding interferes greatly with the procedure, but with patience this can be controlled. The patient suffers some pain, especially when the sac is reached, but I always have been able to finish the operation under local anesthesia. During the following four to six weeks the patient must be kept under careful observation, and excessive granulations around the opening must be removed. This is an important point, because the opening tends to close or to become partially closed during the healing process. If at the end of six to eight weeks the edges of the opening are firm and smooth, the case may be considered cured.

The twenty cases referred to in this paper include all the cases I have operated upon up to January 1, 1917. I have not included cases done in 1917, because it might be said that no opinion as to the final results could be formed. Naturally the early cases were not so well done as the later ones. All of these patients were relieved of suppuration at once, and all with whom I have been able to keep in touch have remained free from suppuration. There are several factors entering into the question of tearing. In the first place, the opening into the sac must be kept clear. I have been able to accomplish this in all cases where the opening was made large enough at the time of operation and the patients have been faithful in returning for treatments. Of course, the canaliculi must be open. Many of these cases have had the canaliculi injured by previous treatments, and in such cases the results are not so good as in cases where the canaliculi are in good condition. A slit canaliculus does not work so well as an intact one.

Ten of the twenty cases have remained free from suppuration and tearing for more than a year. Four cases operated upon in the latter part of 1916 are well. Three cannot be traced, but were well when last seen. Three cases complain of tearing at times. In one of these, the opening into the nose was not made large enough, and while it did not close completely the edges of the opening were too close together, and probably the aperture was clogged at times. One case had the lower canaliculus completely closed as a result of previous treatment, and the upper canaliculus was injured also. This case was relieved of suppuration, but there was tearing. The third case with tearing was that of an old lady with drooping lids. The lower canaliculus had been slit and a small opening had been made into the nose. This opening was closed at the time of my operation. Pus was running over the face from a fistula into the sac. The patient was relieved of suppuration, but has tearing at times. One case with a slit lower canaliculus has no tearing unless he holds his head forward for some time.

The case of Miss E. N. is interesting. In 1913 she had the right sac removed, with an excellent result, except that tearing bothered her. In May, 1915, I did the intranasal operation on the left side, which had been suppurating for two years. She was relieved at once and has remained well. The left side does not tear, and she says that she gladly would have the same operation done on the right side if it were possible.

In this case the canaliculi were in good condition and the opening has remained clear.

CONCLUSIONS.

The results of the operation are ideal if the canaliculi are in good condition, the operation well done and after-treatments carefully carried out.

Suppuration is relieved invariably.

Even in cases with defective canaliculi, there is little tearing, and then only at times, or with certain positions of the head.

The opening into the sac must be made large with smooth edges, and the edges must be kept smooth till healing is complete.

104 S. Michigan Ave.

LXIII.

REPORTS OF MASTOID CASES FROM AN X-RAY STANDPOINT.

By George Sloan Dixon, M. D.,

New York.

Gentlemen—Your Chairman has done me the honor to ask for the exhibition of some X-ray plates of the mastoid exposed in the course of our work at the New York Eye and Ear Infirmary, and report the X-ray and pathologic findings. It gaves me great pleasure to comply.

5837.—Miss E. J., about nineteen years of age, a case of Dr. Dench's. Acute suppurative otitis media, left. The same side had been affected some years previously, and whether the mastoid should be explored depended upon the X-ray findings.

She was examined September 28, 1917. A clear pneumatic mastoid was found on the right side.

The left mastoid was a hazy to cloudy pneumatic, but there was no evidence of breaking down, and it was not cloudy enough to indicate the necessity for operation. So it was reported: "Not operative at present." The case cleared up without operative interference.

5965.—Mr. G. Admitted to the infirmary October 28, 1917, in the service of Dr. Bacon.

This case had been referred to me by an outside surgeon on the 26th of October, and the following report was made:

"Has a very large, clear pneumatic mastoid on the right side. Sinus about the usual position.

"Left side has apparently also been of pneumatic type, but it is thoroughly disorganized. There is no question as to its being an operative case, and think that a large perisinus abscess and probably an epidural will be found."

He gave a history of sea bathing in July; complained of pain in the left ear shortly after, for which he attended a dispensary. There was slight discharge. Swelling appeared behind the ear in the latter part of September. In a few days this swelling subsided, but reappeared last week—that is, the week previous to October 28th. He complained of pain and headache.

The case was operated on October 30th, two days after entering. Pus and granulation tissue were found under the cortex. He had a perisinus abscess at the knee, and the sinus was covered with granulations. A zygomatic abscess was also present. He is still in the hospital, but doing well.

5721.—L. D., aged twenty-six years, was admitted to the infirmary August 30, 1917, in the service of Dr. Lewis, with a history as follows:

"Four weeks previously he developed an earache on left side, following a swim. He fainted the next day. For five days earache continued, until it began to discharge, and the discharge has continued ever since."

Examination showed the canal filled with foul pus; pouting tit-like granulation protruding from superior portion of membrana tympani; faint, deep tenderness over the antrum.

He was X-rayed on the 28th of August—two days previous to admission.

The right mastoid proved to be a well developed, clear pneumatic, with sinus about as usual.

The left mastoid was also of a pneumatic type, very cloudy throughout; sinus forward at knee, with fairly clear evidence of loss of bone over and just below the knee. Perisinus abscess suspected.

A mastoidectomy was done by Dr. Blackwell on the 30th of August, who reported that he found no free pus. There was a large amount of granulation tissue about the antrum over and above the knee of the sinus, with trebeculæ broken down.

There was no free pus in this case, but the fact that the trebeculæ over the knee having been found broken down was sufficient to warrant the X-ray reading.

5865.—Mrs. M. E., aged forty-five years. A private case of Dr. Dann's.

Dr. Dann referred the case to me on the 4th of October. I reported both sides as fairly well developed pneumatic mastoids, there being very little difference between them, neither being particularly clear. The right was a trifle hazier than the left, and the septa a little less distinct.

There was a peculiar little area just above the antrum of the left side, which seemed to be in the squama. It was circumscribed, about three-eighths of an inch in diameter, and gave the impression of a sequestrum; the short duration of the case was against this.

She did not do well, and returned on the 9th for reëxamination, when the plate showed that the area above the left antrum had changed its appearance, what seemed to be a sequestrum having been partially absorbed. It was then found to be a case of streptococcus mucosus capsulatus infection, and a diagnosis of probable epidural abscess was made.

Dr. Dann operated the next day and found the bone badly disorganized, with an epidural above, as reported.

5848.—Mr. K., aged forty-two years. A private case of Dr. Lewis. Was admitted to the infirmary on the 1st of October, 1917.

Dr. Lewis informs me that the patient had been complaining for about five weeks before he saw him. There was slight discharge; no tenderness; no temperature, and very few canal symptoms. There was a little drooping of the posterior-superior canal wall, but it was not marked, and there was a tit-like perforation of the membrana tympani. He said that it was a case which he thought might be operative, but he did not care to undertake the operation without evidence which an X-ray examination might supply.

X-ray examination was made on October 1, when it was found that the left was a clear pneumatic bone, with a sclerosed postsinus area and tip.

The right was found to be an exceedingly cloudy and apparently disorganized pneumatic, with sinus well back, and operation was recommended.

Dr. Lewis operated the same day, found the entire bone softened throughout, and the sinus and dura exposed by disease. Patient made a good recovery.

5853.—Miss J. W., aged twenty-nine years. A private case of Dr. Saunders.

Was referred for X-ray examination on October 1st. The right mastoid was found to be fairly well developed and of pneumatic type, the sinus being located about as usual.

The left was a very cloudy, apparently pneumatic bone, with sinus somewhat superficial, but otherwise in about its usual location. The appearance was so unusual that the patient was asked to return. She did so on the 3rd, and the findings of the former plate were confirmed, and operation suggested.

She was admitted to the infirmary on the 5th of October, and Dr. Saunders operated the same day. The entire mastoid was filled with pus, and most of the cells had necrosed. The pus was under pressure; a perisinus abscess exposed a small area of sinus just below the knee. She made a good recovery.

A smear from the mastoid pus showed that it had been a case of streptococcus mucosus capsulatus infection.

I have little information as to the history of the case, but she was going about, and was apparently in very fair general condition. Dr. Saunders will probably have something to add.

5827.—V. DeL., aged sixty-five years. Was admitted to the infirmary, in the service of Dr. Whiting, with a subperiosteal abscess following a right otitis media purulenta chronica. The infection was streptococcus mucosus capsulatus of six weeks' duration, and of course would have been operated on without an X-ray examination, but it was done in the routine course of our work.

His left mastoid was found to be a pneumatic bone, with a sclerosed-looking lower half, the sinus being about the usual position.

The right—a very cloudy pneumatic; the sinus not shown. A large area of soft bone from the antrum back and below, measuring one inch vertically by one and a half anteroposteriorly.

Diagnosis of epidural and perisinus abscesses made, and operation of course recommended.

A mastoidectomy was done by Dr. Dann on the 24th of September, who stated that he had never seen such extensive destruction, and that the X-ray findings were fully confirmed.

6004.—Mrs. W. F., aged sixty-two years. Service of Dr. Lewis. She presented herself at the infirmary for treatment of her right ear. She had practically no mastoid tenderness. She had what looked like a furuncle on the posterior canal wall, well outward, which obstructed the view of the fundus.

At this time there was no purulent discharge. It was serous. Her hearing on this side was good. She could hear a low whisper at several feet. Shortly after this the discharge became purulent, and she had more tenderness. She could give no correct idea as to the length of time the ear had been involved. She said it was two, three, or four weeks, and she gave no history of marked pain in the ear.

X-ray examination was made on November 3rd. The left mastoid was found to be a clear pneumatic. The right had the appearance of being completely disorganized; sinus in about the usual position.

At the operation the cortex was found fairly thick. There was marked breaking down of the bone throughout, and a perisinus abscess was found.

December 14, 1917.

LXIV.

DENTIGEROUS CYSTS, WITH REPORT OF A CASE.

By Ira Frank, M. D.,

Chicago.

Before proceeding to the history of the patient, allow me briefly to state certain facts regarding cysts of the jaws. Maxillary and mandibular cysts have long been known and were recognized by ancient medical writers, but it was not until the first quarter of the nineteenth century that records show an attempt to differentiate the various types of hollow jaw tumors, and any serious effort to account for them etiologically. Magintot, in 1872, was the first to attribute cysts of the jaw to embryonic rests of dental tissue. In 1885 Malessez, in France, published his theory of the development of dental cysts; and further study by numerous observers and students has added little or nothing to his contribution. According to the Malessez theory, cystic odontomas arise from a small irregular cell mass about the roots of the teeth, called by him "débris epitheliaux paradentaires." This débris, which is the remains of the embryonic dental ridge, the epithelial cord, and the outer layer of the enamel organ, is found in the jaws of adults throughout life.

Probably the simplest classification of cystic odontomas is that recently suggested by New, of Rochester. I have been prejudiced in favor of this classification for the simple reason that it establishes a direct simple nomenclature, which is something no two previous writers seem to agree on. New recognizes two classes of cysts, namely: (1) Simple cysts, and (2) adamantinomas. The first or simple group consists of dental or root cysts, and secondly, dentigerous cysts. The second group contains the enamel odontomas and the malignant multilocular cystic tumors.

The simple dental cyst is the most common of the cystic odontomas, and arises from the paradental débris through abnormal irritation or stimulation. The irritation may be the eruption of a tooth or some form of peridental inflammation, such as alveolar abscess or the dead root of unfilled teeth

They may, therefore, occur at any age, and cases reported show an age range from early childhood to seventy years. The tumor is said to vary from the size of a cherry to that of a small orange. They do occur much larger; and to continue in the vegetable system of measurement, Brophy records a cyst as large as a fair sized cantaloupe.

The walls of the cavity are composed from within outward of thin flattened epithelial cells and a layer of connective tissue and bone which varies in thickness according to the size of the cysts. When large, the growth erodes the surrounding bone, which gradually thins and separates into fragments to form the socalled eggshell crackling as a clinical symptom. The contents of the cavity are a thin white or yellow fluid, sometimes mucoid in character, with broken down epithelial cells and cholesterine crystals floating in it.

The dentigerous or tooth bearing cysts are believed to arise from the Malessez débris most generally, though Bland-Sutton is of the opinion (and Brophy coincides with this) that they are formed from the epithelium which dips down into the submucous tissue to form the enamel organs; in other words, an expanded dental follicle. These cysts occur less commonly than the simple dental cysts, and are usually located in the bicuspid or molar region in either jaw. They form and grow principally during or shortly after second dentition and therefore occur early, except when they appear in association with the eruption of the third molar or wisdom teeth. They may, however, not be noticed until late in life.

Characteristic of this type is the absence of a tooth corresponding to the position of the growth, and, possibly, an irregularity in the two teeth adjoining the vacant space. The swelling may grow to great size as in the simple dental cysts, and limit the motion of the jaws. It may extend outward hideously to distort the face, or, in the superior maxilla, erode into the antrum. The cyst wall is similar to that of the dental cyst, but the contents differ in that the missing tooth is found imbedded quite loosely in the substance of the wall. This tooth, and more rarely the remnants of a number of malformed teeth, are seldom developed—especially is this true of the roots.

The following history and findings, the diagnosis of which was a simple matter with the use of the X-ray, is fairly typical, differing only from the general run of recorded cases in minor details.

The history of the case is briefly as follows: Male, twelve and one-half years of age. When six years of age parents noticed a swelling in upper jaw just beneath the nasolabial fold on the left side. The growth has at no time been painful and has increased in size very slowly until at the time of observation it was about the size of a small plum. The lateral incisor, cuspid, and bicuspid teeth on the same side were missing and have never erupted. The mucous membrane appeared normal, no glandular enlargement was present and palpation elicited a parchment-like crepitation.

Nasal examination negative. A roentgenogram demonstrated the true character of the tumor, showing distinctly a cyst with the three missing teeth present (Figure 1). At the time of operation the tumor had assumed such proportions as to entirely fill the cavity of the antrum of Highmore. It was removed by making an incision on the buccal surface of the superior maxilla similar to that employed in performing the radical antrum operation. After exposing the mass it was incised, allowing the escape of fluid and revealing the location of the teeth, which were firmly imbedded in the alveolar process. The cyst (Figure 2) was easily shelled out, after which the wound was closed with the exception of a small drain. Recovery uneventful.

Pathologic Report.—The cyst measures three by three by two centimeters. Outer wall is whitish and smooth. The inner surface is pink in color. The wall is three millimeters thick. There are three rather small but well formed teeth.

Microscopic Description.—The wall of the cyst is formed by dense fibrous tissue, many of the bundles of which are homogenous and hyalin. Many small, vacuole-like spaces, apparently due to edema, are present. The tissue contains moderate numbers of thick walled blood vessels, the walls of which are markedly hyalin. Wide, thin walled blood vessels, lined by prominent endothelium, are somewhat more numerous. Scattered diffusely throughout the dense tissue of the outer half of the wall are moderate numbers of lymphocytes; about some of the blood vessels these are grouped to form dense foci. In the inner half of the wall the lymphocytic infiltration, diffuse as well as perivascular, is very much greater The inner surface of the cyst is lined by a layer of squamous epithelium, of about the same thickness as that of the normal buccal mucosa. The epithelium lies upon a row of short, regular papillæ, from which the epithelium is very sharply defined. The thin walled vessels of the papillæ are distended with blood. The epithelium is richly infiltrated with polymorphonuclear leucocytes, smaller numbers of which are intermingled with the lymphocytes present in the stroma of the papillary layer.

Diagnosis.—Dentigerous cyst, with acute and subacute inflammation.

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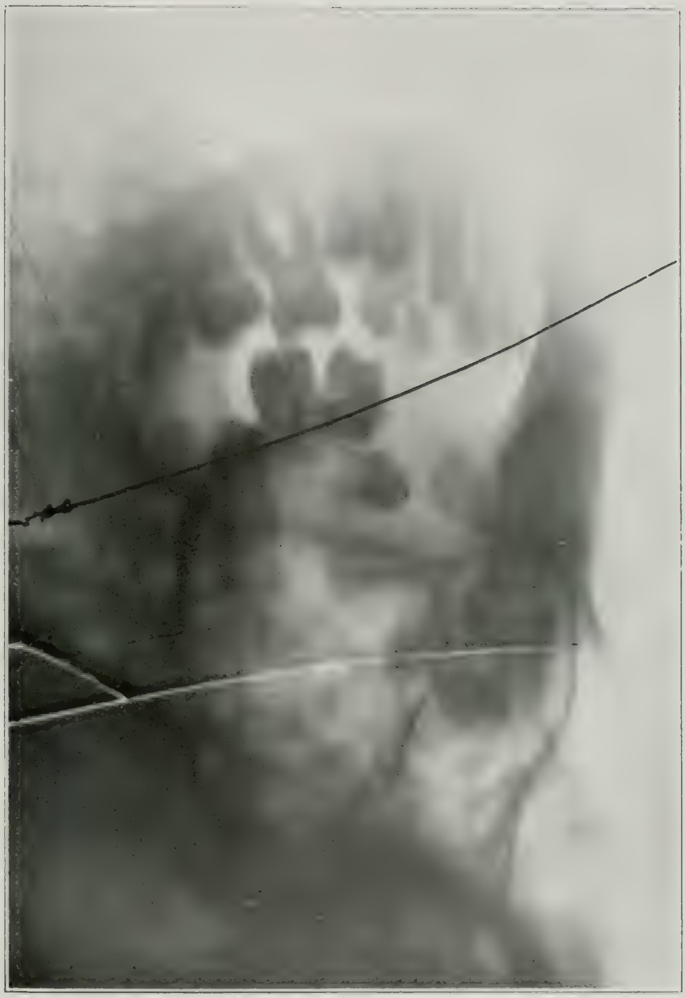

Figure 1.—(Imperfection due to crack in plate.)

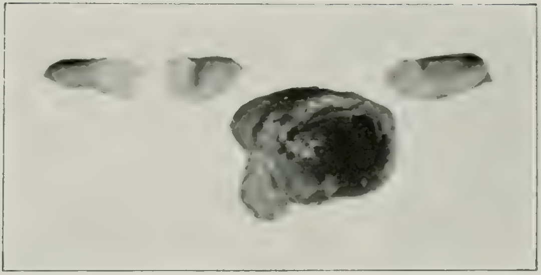

Figure 2.—(About seven-tenths actual size.)

LXV.

CLINICAL OBSERVATIONS ON SUBACUTE MASTOIDITIS.

By Hugh B. Blackwell, M. D.,

New York.

The expression, subacute inflammation, as applied to the mastoid, is here used in its broadest sense. It is meant to indicate that period of mastoid inflammation which follows immediately on the disappearance of all acute inflammatory phenomena, and its duration extends until the patient has made a complete recovery from the original infection or a chronic suppuration has been established.

The term "resolving mastoiditis" is often applied to this condition, but its use would suggest that the patient was improving. For this reason, the author believes that the appellation is misleading, carelessly applied, and its use is often based on a limited appreciation of the potential gravity of these cases.

Duration.—The acute period of mastoiditis might be said to cover anywhere from a few days to a week or more. As the acute bone tenderness disappears, earache and throbbing cease, the temperature falls, and the condition might be said to become subacute, without the quantity or quality of the aural discharge having undergone any alteration. A subacute inflammation having existed from two to three months, it arbitrarily becomes one of chronic suppuration.

Pain.—Objective pain, such as mastoid tenderness, is not a very conspicuous symptom in subacute mastoiditis. In fact, it is usually conspicuous by its absence or by being noted only in deep pressure. Generally speaking, the longer the duration of these cases, the less evident is the bone tenderness. This should not surprise us when we consider the continuous structure of all mastoid cells with each other and the middle ear. During subacute inflammation this anatomic condition easily permits the establishment of free drainage from the mastoid cells, thus relieving the tension and incidentally the pain. Furthermore, these structural characteristics render it possible for a mastoid to undergo marked degeneration during the subacute stage, and yet present but little or no pressure tenderness.

Subjective pain is a variable factor. Headache is much more constantly present than during the acute stage. It is chiefly nocturnal and usually intermittent. Discharge is the least constant factor in subacute mastoiditis. It may be most profuse in quantity, of heavy thick consistency, appearing through a drum perforation in a pulsating manner. On the other hand, it may be entirely absent, or it may exist as a thin, scanty, mucous or serous exudate.

Audition.—The impairment of aural function is subject to wide differences, but usually in cases requiring operation the hearing in the affected ear is very much lowered.

External Auditory Canal.—Fundus changes in the canal may show the greatest variations. The drum may present all the evidences of a severe mastoid suppuration, or, on the other hand, it may appear to the uninitiated almost normal. Between these two extreme pictures there may be present almost any degree of inflammatory reaction.

The cause of premature middle ear resolution in subacute mastoiditis is the formation of a thick plug of organized granulation tissue in the antrum, which tightly seals it and completely separates the middle ear from the mastoid, thus permitting the middle ear to assume a more or less normal appearance and allowing the mastoid suppuration to continue undrained until it either ruptures spontaneously again into the tympanum and reestablishes the aural discharge or terminates in the solution of a table of the skull, or a spontaneous cure.

I have had numerous cases of this sort. On opening the mastoid the cells may be found to be extensively diseased; but the antrum, instead of being broken down and represented by a space filled with pus and loose granulations freely communicating with the mastoid, is found to be dry and tightly filled with this thick plug of organized tissue. The development of this condition is of course favored by the small size of the antrum with its thick bony walls, and probably accounts for the large number of cases that are reported with normal drums associated with suppurative mastoids. I have never seen one of these cases where the drum was absolutely normal. They always had some appearance of semiresolution, and the patients have always had some subjective disturbance. For this reason, I wish to emphasize the importance of recognizing and appreciating the value of all clinical appearances, however slight.

There is still another condition that I have noted, namely, where the inflammation presents or ruptures in the posterior bony canal wall, giving all the appearance of a furuncle of the posterior wall of the external auditory canal. In several of these conditions the furuncular swelling has departed from the usual type in that it was cone-shaped, coming to a point and sloping away evenly in every direction. From the apex of the swelling the pus discharged drop by drop. Behind this appearance could be seen a resolved drum. At the operation, on lifting the membranous from the bony wall just underneath the swelling, there was found a mass of granulations protruding evenly from a small round bony cortical perforation, which elevated the overlying membranous wall and produced the condition.

Operative Indications.—One naturally hesitates to speak at length on this point to an audience so thoroughly versed on this subject, but since many cases of this class vary widely in their clinical appearances from each other and from the orthodox, well established and framed picture of acute mastoiditis with clear operative indications, a few words on this phase of subacute mastoiditis may be well taken.

A situation that frequently confronts an otologist in the treatment of mastoiditis is as follows: The acute stage having passed, the discharge having become much reduced or perhaps entirely ceased, mastoid tenderness almost or entirely gone, and the earache disappeared, one would naturally regard the case as much better were it not for the fact that the tongue is still coated, bad breath present and some nocturnal headache continuing. The patient and his friends are inclined to regard his condition as much improved, and it is frequently somewhat of a shock for them to realize that this feeling is not shared by the otologist. Under these circumstances, their questions are a source of embarrassment to the physician, who puts off from day to day the giving of a definite expression of opinion, during which time he is looking and hoping for a marked local change in the patient's condition that would either render an operation imperative or point the way to a spontaneous cure. Should the mastoid happen to be well broken down, an X-ray would be of great assistance in definitely clearing up the question of the necessity for an operation. Unfortunately, however, there are many of these cases in which there is only a slight pathologic change in the bone of the mastoid. This change is not sufficient to register definite signs of bone destruction, so that a plate of such cases would show a mastoid apparently only slightly diseased, or almost normal. These mastoids, while innocent from an X-ray standpoint, are capable of producing the gravest intracranial complications. Accordingly, we are thrown back to a consideration of the clinical evidence presented in determining the necessity for an operation.

Now the question is, What clinical evidence should cause us to advise an operation where the results of the X-ray examination are more or less negative, the canal being dry or almost so, with a drum of resolving appearance, and little or no mastoid tenderness present? Briefly, it is as follows:

1. History.—A history of four weeks' duration; nocturnal pain or restlessness; queer feeling in the head; any slight temperature not attributable to other causes; deep bone tenderness only determind by comparison with the normal side, and lastly, but most important, evidence of general toxic absorption, as shown by coated tongue, bad breath, headache and lassitude.

It has been my own observation that when an operation is really indicated these patients generally sense the gravity of their own condition and readily assent to any suggestion in favor of mastoidectomy. Some who subsequently develop meningitis seem to be stimulated to intermittent spells of great activity, probably caused by early cortical irritation. I have had one such patient. We must remember that all streptococcus infections are more serious than other forms. I have had a number of capsulatus infections recover without operation.

Finally, in regard to the value of the X-ray as a diagnostic aid in determining the necessity for an operation, I wish to say that I believe it to be of great assistance when the plates are taken by one so thoroughly versed in the subject as Dr. Dixon. However, the findings must always be weighed in conjunction with the clinical evidence, which, after all, is our main dependence in reaching a definite conclusion.

148 West Fifty-eighth Street.

LXVI.

NOTES ON CASE OF MENINGITIS OF OTITIC ORIGIN.*

By Dr. Alfred Kahn, M. D.,

New York.

On July 23, 1917, Mr. A. N., twenty years of age, presented himself at the New York Eye and Ear Infirmary on the service of Dr. Dench. He complained of headache, which he localized over the right side of the head, having had this headache off and on for a period of several weeks. He had felt dizzy for about the same period of time at intervals. His general condition was good; temperature 99°. The fundus of his right ear showed quiescent otitis media purulenta chronica. There were no signs of an acute process. He stated that he had had a discharging ear off and on for a period of about four years. At the time of examination, the external canal did not contain any discharge. He did not have spontaneous nystagmus. The static labyrinth tests were normal. On account of his dizziness and headache, he was admitted to the ward for observation, with a diagnosis of possible cerebellar abscess. His condition remained unchanged from date of admission, his temperature ranging between 99° and 101½°, until July 29, when he suddenly developed a rapid rise of temperature to 106°, with very slight mastoid tenderness. A diagnosis was then made of episinal abscess with possible jugular involvement. The mastoid was opened, an epidural abscess, with clot in the sinus, found. A radical mastoid operation, with removal of internal jugular vein, was performed. July 30th, the next day, his temperature was down to 100°; his temperature curve gradually dropping, ranging between 98½° and 100½°, until on August 4th, about seven days after the first operation, his temperature again suddenly rose to 104°. The patient became delirious and showed symptoms of meningitis. He was taken to the operating room, the mastoid wound explored, a large area of dura exposed—but the dura was not incised. (It is my opinion that if it had been incised the patient would have died.) The sinus was again opened and allowed to bleed freely from above. The lower end, of course, was tied off at the first operation. I believe that it was this procedure of freely bleeding, thereby lowering intercranial pressure, that caused a cure in this case, and it is further my belief that it is a valuable suggestion in the treatment of meningitis of otitic origin. It seems superior to exposing the subdural space in the presence of a meningeal infection for the purpose of decompression. After the second operation the patient's temperature rapidly dropped to 100°, gradually vibrating to normal, until, on August 31st, he was discharged cured.

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*Read before the New York Academy of Medicine, Section on Otology, December 14, 1917.

Considering the case more in detail, his recovery would seem to be due, first, to the prompt radical mastoid and jugular operation, and, in the second operation, to the bleeding when the lateral sinus was opened, and to frequent lumbar punctures. In this connection it occurs that the procedure of opening the lateral sinus for the relief of brain tension in meningitis is a good one. This may especially be true in those cases where the meninges are infected by way of the internal ear. It is a known fact that pressure made over an exposed lateral sinus will indirectly increase the rise of endolymph in the modolius. If the modolius is broken, pressure of the sinus will increase the escape of endolymph. It is also known that pressure over the cerebellum will increase the irritability of the labyrinth. This is probably due to increased tension indirectly on the endolymph in the semicircular canals.

Having this experiment in view, it occurs, as a matter of fact that, conversely, by opening the lateral sinus and bleeding directly from the brain tissue, brain tension will be reduced. For this reason, it would seem that the recovery of this man might be due to the fact that the lateral sinus was freely bled. Along the same line, spinal punctures probably also aided in keeping his brain tension low. This procedure has been mentioned in the literature on this subject before, especially by Dr. Dench in a paper read before the International Medical Congress in Buda-Pest several years ago.

Considering the case further, it is of interest apparently at first from the lack of diagnostic symptoms. There was no mastoid tenderness, no bulging of the external auditory canal. The fundus did not show any active process. It was quiescent. On account of his severe headache and dizziness, he was admitted as a case of possible cerebellar abscess. The first operating indication was the rapid rise in temperature, accompanied by chill, with loss of consciousness. A diagnosis of episinal abscess with possible jugular involvement was then promptly made, and on opening, such was found to be the case. Following the operation there was an immediate subsidence of symptoms and returned consciousness, followed in one week by meningitis, after the patient was apparently well on the road to recovery.

BLOOD SIGNS AND LABORATORY FINDINGS.

The laboratory findings (blood, spinal fluid, etc.) and readings are of such marked interest that the case would be incomplete without reporting them. On July 25th, four days before first operation, the blood showed 15,000 white cells, of which 57.8 per cent were polymorphonuclear leucocytes. On August 4th, ten days later and six days after the first operation, the blood showed an increase of polys to 92 per cent; the question might arise was this increase of polys due to infection of the sinus between the torcular and the mastoid opening, or was it due to meningitis? On that day, blood culture, after seventeen hours, was negative; but on the same date the spinal fluid contained cells equal to pus. This is indicative and is diagnostic of meningitis. On August 6th, two days following the second operation, the spinal fluid was less turbid, and the white cells were reduced from an uncountable pus equality to a cell count of 2,480. The globulins were reduced from a count of four plus to two plus. Fehlings was negative. Culture was negative. On this date the temperature was dropping and the patient returning to a normal state, showing that the second operation was exactly at the turning point towards recovery.

Finally, in closing this report, let me state that I do not know of any case in the literature of meningitis where there was a spinal fluid cell report equal to pus and where the patient lived.

50 East 42d St.

LXVII.

CHANGING METHODS AND ADVANCES IN THE TREATMENT OF PROGRESSIVE DEAFNESS FROM CHRONIC SECRETORY OTITIS MEDIA.*

By Francis P. Emerson, M. D.,

Boston.

For the reason that otology is not an exact science, there are problems connected with its practice that should be reconsidered at stated intervals. One of these is the diagnosis, prognosis and treatment of that form of progressive deafness which is the result of chronic secretory otitis media.

Previous papers on this subject were read before this society by Dench in 1894, Harris in 1908, and by Holmes in 1912. These three papers are the only ones of record, with the exception of Reik's paper on the relation of the tonsils to middle ear deafness.

The discovery of lymphoid hypertrophy in the vault of the pharynx, and the recognition of its relation to the eustachian tube by Wilhelm Meyer, marked an epoch in the prevention and treatment of this form of middle ear disease. This was fifty years ago. Following this discovery, the further appreciation of the close relationship of the nasopharynx to the middle ear made the organization of this society a logical step. A large proportion of its present membership were educated first as otologists, and they have since studied and treated the nasopharynx only so far as in their judgment such treatment, operative or nonoperative, could influence the prognosis of progressive middle ear disease.

In consulting the files of this society, we find frequent reference made to the necessity of stopping infection by way of the eustachian tube. Hardly a writer, with the exceptions noted, has ventured to clearly define what he considered to be indicated along this line. The teaching in our universities makes no attempt to correlate the two subjects of rhinology and otology in such a way that the student should know what to do, and what not to do rhinologically, to influence the middle ear. Each student is left to work out his own clinical experience, beyond instruction which relates specifically to the vault of the pharynx. Observation among the leading otologists of today would lead us to infer that we are divided into two classes in our efforts to protect the middle ear. In the first class are those whose treatment is almost wholly confined to the nasopharynx and Rosenmüller's fossæ, to such an extent that it is common practice for their patients to consult them as aurists, and their confreres, as rhinologists. In the second class are those who correct all deformities of the septum, abnormal conditions of the turbinates; drain the sinuses, remove tonsils, etc., and in fact do what any rhinologist would do, and believe that in so doing they retard or check the progress of middle ear disease.

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*Read before the American Laryngological, Rhinological and Otological Society, Atlantic City, May 31, 1917.

The writers of our latest textbooks give very scanty reference to remote foci, in discussing the etiology of chronic secretory otitis media.

In order that the writer might not misconstrue the attitude of the first group, namely, those who lay especial emphasis on the nasopharynx, he sent the following question to fifteen prominent aurists:

"Given a case of progressive deafness as the result of chronic secretory otitis media, where the hearing for the whispered voice has been reduced to 15/25ths, or less, would you expect any improvement from treatment of the nasopharynx, excluding the epipharynx and inflation?"

Replies were received from ten, all of whom said they would not expect any benefit, provided the lymphoid tissue in the vault and fossæ had been thoroughly removed. One considered such a case as a beginning otosclerosis, and one had seen active operative measures do harm. In order to prove that positive improvement in hearing results from the cure of remote foci is not an easy problem, as the testimony of the patient or of the aurist is of no scientific value, unless such observations are supported by careful hearing tests and records covering a term of years, for we are dealing with a chronic disease which is subject to acute exacerbations and varying acuity of hearing.

Further, in order that such records should be available for analysis, such observations should extend over a period of from three to five years.

It would seem appropriate, after this length of time, to consider again whether we have made any advance in the treatment of chronic deafness over the pioneer work of Meyer and Toynbee fifty years ago. Their work has since been elaborated by Politzer, Lucae, Siebenmann and others, but the essential facts are unchanged. Can we today offer any more hope to this unfortunate class of patients who comprise such a large part of our clientele? If some of them can be helped, is it possible to predict as a result of examination, that good results will follow our treatment in a given case, and how far any lost hearing may be restored?

In order that we may discuss this subject from common premises, let us define what we mean by chronic secretory otitis media. In a given case, otoscopic examination would show a dull gray or ground glass appearance of the membrana typmpani, absence of the light reflex, and retraction. The clinical picture would be one of slowly progressive loss of hearing, usually more advanced in one ear, with or without tinnitus. The hearing test would show a positive Rinné, the Weber lateralizing toward the more deaf ear. The low note by the Dench fork is unchanged, or slightly raised. The bone conduction is unchanged. The whispered voice, however, may be reduced to as low as 2 to 15/25ths. Given such a case, what conditions, if any, outside of the treatment of the epipharynx, will influence its slowly progressive course? Whether any of the lost hearing can be restored is of the greatest importance to the patient, for the restoration of one or two notes in the upper register will determine to a marked degree the efficiency of the individual.

In order that we may definitely promise ourselves or the patient that the hearing will improve, the writer is guided by the following considerations, all of which depend on the question of reinfection.

It is his experience that in every case of progressive deafness from chronic secretory otitis media, there is a primary focus which is constant for that individual. This may become quiescent, but in the interval it remains as a low grade process, subject to acute exacerbations. This point I wish to emphasize, for until the primary focus is drained and cured, such acute exacerbations are a constant menace to the eustachian tube and middle ear. The course of the disease will be from bad to worse, and a little more rapidly if inflation is practiced as a routine.

That there are pathogenic bacteria in the mouth and pharynx at all times is common knowledge. That they possess sufficient virulency under favorable conditions to cause serious pathologic conditions in remote organs has been proven by the investigations of Miller, William Hunter, Billings and many other observers.

Depending upon the organism and the virulency of the infection, the clinical picture is one of toxemia, septicemia or pyemia running an acute or chronic course. While this work is of recent date, all such facts had been observed years ago by Professor Arkovy of Budapest, from 1878 to 1898, substantiated by the painstaking microscopic findings of Miller of Berlin, from 1884 to 1894, and by the clinical experience of William Hunter of London, who published his results in the Practitioner in 1900. Miller, who was educated as a physician and also as a dentist, showed bacteriologically that focal processes in the teeth, tonsils, or sinuses kept up a low grade of infection of the adjacent tissues, and during exacerbations this might extend by continuity, or directly by way of the lymphatics or blood stream, to neighboring or remote organs. That such low grade infection is constantly present, either in the nasopharynx or quite as often at some other point in all cases of progressive chronic secretory otitis media, has been the observation of the writer for many years.

In order to determine where this primary focus may be, we inquire of the patient, in taking the history, where these acute exacerbations begin. It is a well known clinical fact that patients subject to acute infections almost invariably claim that for years such infections commenced in a certain manner—i. e., as a sore throat, and then went up or down, or started as a head cold, and then went down.

In a given individual this onset is almost constant, and does not usually alternate over many years, and indicates that the primary focus does not entirely subside, but becomes quiescent and is subject to acute exacerbations whenever the resistance of the host becomes lowered. This, then, gives us our first clue as to where we shall begin our treatment, and cannot be determined by the anatomic conditions of the nares, even if a rhinitis be present. The next question is to ascertain if the hearing varies—i. e., whether it is better on some days than others. In such cases an active cause of the otitis is still present outside of the middle ear, and we can usually hope for improvement up to their best hearing, or more, if we remove the original focus and clear up the accompanying infection.

Let us suppose a case where the history points to the acute exacerbations being more or less constant in the nose. It is the writer's opinion that no amount of anatomic deformity of the septum influences the course of chronic secretory otitis media until after infection takes place, but that such deformity predisposes to infection.

The tendency to a low grade infection in the nares is promoted by the following conditions: Given asymmetrical nares from deviation of the septum, there results a compensatory hypertrophy of the turbinate on the concave side from overfunction from nature's attempt to equalize the air current. As a result, the soft tissue goes through the various phases of congestion, increased connective tissue and thickening of the mucosa. At this stage any acute infection on account of defective drainage may be continued as a low grade process subject to acute exacerbations. With the defective drainage, if a new infection is virulent enough, the sinuses may become involved. We therefore operate in the nares, not to remove a mechanical obstruction per se, but to improve drainage and clear up the attending infection.

To one who accepts this pathology it is essential to the integrity of the tube and middle ear that the infections of the nasopharynx should be cured so that acute exacerbations may be prevented. This can only be accomplished by improving the defective drainage and equalizing the air pressure by reconstruction of the nasal chambers—i. e., straightening deflected septa, removing redundant tissue from the turbinates, reducing posterior hypertrophies, and opening all infected cells.

After this it is equally important in cases of deafness of long standing that no low grade process persists, as a nasopharyngitis, or some focus be left that may still be subject to acute exacerbations, and thus offset the effect of our operative procedures as far as improvement of the eustachian tube is concerned. The middle ear condition grows progressively worse by reason of reinfection from the same primary cause, or from secondary foci, until the hearing is beyond improvement. For this reason auditory reeducation, by whatever means, inflation, electricity, etc., that does not take into account the underlying cause cannot be of permanent value until such focus is removed.

Pharynx.—In the pharynx all lymphoid tissue, however small, should be removed, including bands between the eustachian tube and the pharynx, and degenerate tissue in Rosenmüller's fossæ. Where posterior rhinoscopy shows chronic congestion of the lateral pharyngeal wall, the condition of the fossæ cannot be determined without a digital examination. Chronic pharyngitis indicated by a glazed appearance of the posterior wall, and a change in the secretions, the writer has always found to be due to caries in the sphenoid, either alone or in connection with a pyogenic focus in the posterior ethmoids, and the socalled pharyngitis sicca he has not found to exist as an entity. While the diagnosis cannot be made with an ordinary probe, it usually can be made by using one whose shank is no longer than a knitting needle and that has a fused head on the end.

Tonsils.—The tonsils in their relation to local and systemic disease are only beginning to be appreciated, especially when found in adults between thirty and sixty years of age. As we now understand their pathology they account for many of the diseases that were supposed to be constitutional, such as scrofula, rheumatism, autointoxication, etc. Visual examination is useless. The anterior pillar should be pulled forward, and pressure used between the outer wall and the tonsil. In many cases where the tonsil is an active cause of the deafness, free pus will be demonstrated on one side, usually at the junction of the velar lobe and the middle third of the tonsil, particularly in those cases showing toxemia. This is an enclosed abscess of streptococcus origin and subject to repeated acute exacerbations. The toxemia is marked, and it is probably the cause of chronic nerve degeneration in the labyrinth. Such cases are attended constantly by a low grade pharyngitis. Reik, in his article published in 1908 on degenerate tonsils and middle ear deafness, has fully described their relation to the eustachian tube.

In the writer's experience the extension to the eustachian tube is more often by continuity and through the lymphatics and blood stream along the posterior pillar. The resulting low grade process can be seen not only on the lateral wall, but in the epipharynx and over the surface of the posterior nares, as well as in the mouth of the tube.

Eustachian Tube.—The pathologic processes of the tube in its relation to middle ear deafness, the writer has usually found either at the isthmus or the pharyngeal end. Examination shows the tube, if the process has existed long enough to affect its lumen, as it usually has in chronic cases, more widely open on the side of the most affected ear, hence inflation is not only contraindicated, except for diagnostic purposes, but it makes the loss of hearing more rapid. Indeed, after stopping the infection, one of the next steps in our treatment is to try to restore the lost tone of the pharyngeal end by applications within the tube and by exercises that shall strengthen the function of the palatine muscles. The most experienced aurist will be deceived as to the lumen of the tube by auscultation, as very many cases have a decided narrowing at the isthmus when inflation seems clear. If no mechanical obstruction exists at either end of the tube, nature has provided for sufficient ventilation of the middle ear. If such obstruction does exist in chronic cases, inflation will do no good and will add to the gradually diminishing function of the tubal muscle. Applications within the tube are of great benefit, but they should not be repeated too often, or be strong enough to excite too much reaction. This reaction, where the obstruction has been at the isthmus, has been one of the most difficult things for the writer to control in the treatment of the tube.

The Alveolar Process.—The best work on the pathology of the alveolar process has been done by dentists. The relation of such foci to local and systemic disease has been repeatedly called to your attention by Dr. Haskin. Miller of Berlin, in 1894, showed the seriousness to a patient with low resistance from pulpitis, gangrene of the pulp, pericementitis, alveolar abscess, otitis, osteomyelitis, periostitis, alveolar necrosis and pyorrhea. He called attention to the fact that pyogenic organisms associated with osseous necrosis were very virulent and that the soft tissues over a considerable area showed infection. His bacteriologic findings proved that such local foci might not only involve adjacent tissue, but extend by continuity to the tonsils and pharynx, or through the lymphatics or blood stream to distant organs. For our purposes such diseased conditions should be investigated in conjunction with a dentist, and all devitalized teeth subjected to an X-ray examination, as part of our routine examination in locating foci that may be the cause of a progressive chronic secretory otitis media.

It is often asked why, if such causes are active in the etiology of chronic secretory otitis media, it is not easy to settle the question by producing a series of cases corroborated by hearing tests. The answer is because of the fact that such foci are usually multiple, and few men have systematically attempted to remove all sources of reinfection. Our knowledge of how to drain all the sinuses, of the menace of pyogenic foci in adult tonsils and the alveolar process, is too recent to enable us to tabulate a long series of chronic cases successfully treated. The improvement is necessarily slow and extends over a considerable time. With energetic early treatment there is a more hopeful outlook for this field of otology.

As a line of investigation to be further developed, the writer sees no reason why such active foci as are constantly found in the teeth, tonsils and sinuses should not contain bacteria with selective characteristics—i. e., in some we would have our pathology in the mucous membrane, in others the connective tissue, and in still others the osseous structure.

CASE HISTORIES.

Sinuses. (Focus.)

December 2, 1911. J. F. W., born in New Hampshire, aged fifty-one years, single, provision broker.

Past History.—Measles at twenty-one years of age. Rheumatism three years. Tobacco moderately, no alcohol. Neisser infection. Repeated head colds. Teeth all removed. General health good, except for history of gall stones.

Hearing.—Not normal for ten years. Worse for past eighteen months. Sounds confusing. Sharp pain in both ears. Tinnitus. No history of aural discharge. Acute infections start in the head.

Examination.—Both drums thickened. Light reflex gone. retracted Pus in middle fossa of both nasal cavities. Adenoid tissue central in the vault. Both eustachian tubes open. the right being more open than the left.

Diagnosis.—Chronic secretory otitis media and suppurative ethmoiditis, both sides. Repeated reinfections from a pyogenic focus in the ethmoid sinuses.

Treatment.—Exenteration of both ethmoid labyrinths. Argyrol, twenty per cent, to eustachian tube. Inferior turbinates trimmed. Nasopharynx cleansed with normal salt and sprayed with argyrol, twenty per cent, until infection cleared up. No inflation except for diagnosis.

Hearing Tests.—

December 2, 1911

R.		L.
2/25	W	16/25
2/35	acu.	9/35
30″/10″	R	35″/13″
	+ W	
32	D	32
	(L. L.)	

March 15, 1913

R.		L.
15/25	W	17/25
32	D	32
	(L. L.)	

March 13, 1915

R.		L.
12/25	W	N
30″/10″	R	35″/13″
	W	
32	D	32
	(L. L.)	

May 13, 1916

R.		L.
N	W	N
32	D	32
	(L. L.)	

January 20, 1917

R.		L.
N	W	N
35″/10″	R	45″/10″
	W	
32	D	32
	(L. L.)	

Result.—No colds for a year. No tinnitus. Hearing normal.

CASE HISTORIES.

Tonsil. (Focus).

May 20, 1915. Mrs. H., born in Rhode Island, aged fifty-nine years, married, no children.

Past History.—Scarlet fever and typhoid at twenty-nine years of age. Pneumonia eighteen months ago. La grippe several times, but not severely until 1915. Tonsillitis annually for several years. Acute infections start in the throat. No aural history until five years ago, when the hearing became impaired, beginning in the right ear. Occasional tinnitus. No climatic variations. Slight catarrh. Pyrosis. Ears worse after each throat irritation. Is below weight. Nose red, color poor, and has been a semiinvalid for twenty years. Never rugged. Prominent otologist told her that nothing could be done, and instructed her maid how to inflate the ears daily.

Examination.—Both drums show ground glass appearance, dull, lusterless. Left nares, anterior deviation of septum, so that applicator cannot be passed. Contact with inferior turbinate Middle meatus not injected and no infection present. Septum, sigmoid deflection. Sinuses transilluminate normally; epipharynx negative. Pharynx, low grade pharyngitis. Tonsils, cryptic tonsillar disease. Right, free pus (one or two drops).

Diagnosis.—Chronic secretory otitis media, both sides. Cryptic tonsillar disease. Left anterior deviation of septum. Progressive deafness secondary to tonsillar infection.

Treatment.—Removal of tonsils, and after-treatment until infection was gone. June 2nd, operation, Des Brisay Hospital. Complete tonsil enucleation.

Hearing Tests.—

May 20, 1915

R.		L.
6/25	W	6/25
20″/10″	R	32″/15″
	512F W	
32	D	32

October 18, 1915

R.		L.
4/25	W	20/25
20″/7″	R	25″/11″
32	D	32

December 10, 1915

R.		L.
9/25	W	20/25
30″/15″	R	22″/12″
32	D	32

January 11, 1916

R.		L.
10/25	W	N
32	D	32

Result.—Pyrosis gone, weight increased. The right ear, which was the first involved, gained four feet. The left is normal. No inflation.

CASE HISTORIES.

Tonsils. (Focus).

June 4, 1916. Mrs. A. L. M., born in Massachusetts, aged sixty-six years, widow.

Past History.—No history of aural discharges or otalgia in early life. Hearing has been growing worse for ten years.

Family History.—Patient has two children living. Husband died twenty-one years ago, cause unknown (suicide). One brother and one sister living and in good health. No family history of deafness. Father died aged sixty years, cause unknown. Mother died aged eighty years, cause unknown.

Patient has had some ear trouble for the past ten years, which has slowly been getting worse. Hearing varies and is worse when she is tired or has a cold. History of head colds and sore throats once or twice a year. Ringing tinnitus occasionally. Sensation of stopping of ears. History of rheumatism. Left hand shows swollen joint. Used to wake with a headache. Left tonsil shows cryptic disease. Pulsation in ears since sickness three years ago.

Examination.—Chronic secretory otitis media, both sides. Right tympanic membrane, indrawn and dull. Light reflex gone; no marked thickening. Right tympanic membrane indrawn and lusterless. Light reflex gone. Both tubes open, but the right a little the more. Isthmus not narrowed.

Treatment.—Left tonsillar infection cured by operation. No inflation at any time except for diagnosis. Local applications to tubes. Multiple and slow sinusoidal currents used for five minutes in each ear weekly. Current caused vertigo and nausea at first.

Hearing Tests.—

June 4, 1916

R.		L.
1/25	Whisp.	2/25
10″/7″	R	10″/5″
	256c	
	Weber	
64	L. L.	64
1/10th	U. L.	N

December 15, 1916

R		L.
3/25	Whisp.	3½/25

February 8, 1917

R.		L.
8/25	Whisp.	8/25
N.	U. L.	N.
32	L. L.	32

April 2, 1917

R.		L.
10/25	Whisp.	9/25
13″/7″	R	13″/6″
	Weber	
N.	U. L.	N.
32	L. L.	32

Result.—Patient was gaining the most when she stopped treatment on account of the difficulty of coming into town at her age.

CONCLUSIONS.

1. Every case of chronic progressive middle ear deafness has a primary focus, which persists as a low grade infection, subject to acute exacerbations. In chronic cases such foci are usually multiple.

2. Such primary focus is usually constant for the individual and is indicated by the location of exacerbations.

3. Every case showing variable hearing can usually be improved up to their best hearing, or more.

4. Socalled cases of nerve deafness of nonspecific origin are, in the experience of the writer, due to toxemia from some definite focus.

5. Inflation in chronic cases is unscientific and harmful as a routine, as the tube is already open and has partially lost its tone in the majority of cases, and in those cases not open it does nothing to remove the cause.

6. Nasal obstructions do no harm to the middle ear unless infection is present. Such obstructions, however, are the primary cause in the development of imperfect drainage, which predisposes to infection, and which is always present in cases of chronic secretory otitis media originating in the nose.

7. Foci, whether in the sinuses, tonsils, mandible or epipharynx, are potential factors in the progress of chronic progressive otitis media, either by direct extension or through the lymph and blood streams.

8. No hearing test will forecast the improvement in a given case as long as we have a positive Rinné with variable hearing.

9. Whatever the macroscopic appearance of the membrana tympani, the cause of the deafness is active for a long time outside the middle ear as a toxemia or low grade infection subject to acute exacerbations.

10. Constitutional diseases have but little effect upon the course of chronic secretory otitis media, except to lower the resistance of the patient and make him more susceptible to exacerbations of their localized focus or foci.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.
SECTION ON OTOLOGY.

Meeting of November 9, 1917.

Sinus Thrombosis With Meningeal Symptoms—Operation, Recovery.

Dr. T. Laurence Saunders: John L., aged seventeen years, a truck driver, was admitted to Bellevue Hospital May 13, 1916, with a history of having had a discharging ear for three weeks. For seven days he had suffered with stiff neck and vomiting; headache three days before admission, and chill the previous day.

Physical Examination.—Heart, lungs and abdomen negative; neck moderately stiff; bilateral Kernig; deep reflexes active.

Spinal Tap.—Fluid under tension, but clear.

White blood count, 23,000; polymorphonuclears, 80 per cent.

Urine negative, except for a very faint trace of albumin.

Temperature, 99 to 104 degrees; pulse, 80 to 110; respiration, 28 to 33.

On May 14, he was admitted to the New York Eye and Ear Infirmary, semidelirious and complaining of headache and stiff neck. He had a moderate Kernig, tender mastoid, fluid discharge from the ear; temperature, 104°; pulse, 110; respiration, 24. History of gradually increasing headache for one week, accompanied by chills, vomiting, and fever. History of discharging ear for three weeks.

Noise apparatus showed hearing; no nystagmus; caloric not performed.

Diagnosis of sinus thrombosis was made on history of chills and high temperature.

The vein was resected; it showed no clot and was not collapsed.

The radical mastoid operation revealed a sinus thrombosis and epidural and perisinus abscesses extending almost to the torcular, with foul pus issuing from the sinus. Free bleeding was obtained from both ends.

After operation the patient did badly for a time; temperature, 99 to 104 degrees, and had repeated chills, with delirium and rigid neck.

Six days after operation the patient was out of doors and feeding every two hours, and thereafter continued to show gradual improvement.

May 18th. Headache; lumbar puncture cloudy.

May 22d. Headache better.

May 26th. Still some headache.

Normal course after May 29th.

DISCUSSION.

Dr. Guntzer asked what germ was found.

Dr. Saunders replied that streptococcus was found in the blood culture but nothing in the spinal fluid.

Dr. George S. Dixon said that in cases of meningitis where there is a cloudy fluid and free blood, the globulin reaction will be positive. The sugar may still be present. There may be a high cell count, but unless germs are found in the fluid or cultivated from it these cases nearly always do well. That had been his experience, and he did not believe one was warranted in giving a grave prognosis unless positive cultures were obtained from the fluid, no matter how many cells were found. He had seen one case of pronounced meningitis with as low as 38 cells and another which appeared to be a case of purulent meningitis where there were several thousand cells, but he had come to look upon these cases as favorable if they did not show a positive cultivation from the fluid. It was quite common to have sugar persist, even in the presence of a well marked meningitis.

He had never, however, known a case of streptococcus mucosus capsulatus meningitis to recover. In his experience, this form of infection was invariably fatal.

Paper: Case of Purulent Otitic Meningitis With Unusual Clinical Symptoms and Pathologic Findings.*

By Charles E. Perkins, M. D.

Paper: Case of Hematoma of Old Radical Cavity Causing Persistent Pain.

Dr. John A. Robinson: H. W., aged thirty, was admitted to the New York Eye and Ear Infirmary on the service of Dr. Dench, on October 8, 1917, complaining of intense pain over the right mastoid area. He gave the following history: Some years ago he received a blow on the back of his head. He became unconscious, and so remained for three days. Upon his discharge from the hospital, his hearing was considerably impaired. Five years later he developed an acute purulent otitis, and underwent a simple mastoidectomy. Nine weeks later, for a recurrence, a secondary mastoidectomy was performed. Shortly after this operation he began to complain of persistent aural discharge, postaural swelling, and vertigo. He was again operated upon, a modified radical being done, the outer two-thirds of the posterior wall, the tympanic membrane and the ossicles were left intact.

The patient experienced no further trouble with the ear until the latter part of August, 1917, when he again began to complain of pain over the right mastoid area which was dull and constant. It became very marked in stooping, and caused vertigo with a tendency to fall to the right. For two weeks prior to his admission, the pain had assumed a stabbing character, confined chiefly to the right side of the head and preventing sleep.

Examination showed the old cicatrices slightly thickened; marked tenderness on pressure; pulse, 80; temperature, 99.8°; respiration, 20. A modified radical had been done. The posterior half of the cavity, which was dry, was filled with a bluish, tense swelling, resembling the sinus. A radiogram made by Dr. Dixon showed a suspicious looking area over the knee of the sinus. The patient did not hear the acumeter, but heard a low whisper at one foot.

Both labyrinths responded to caloric and rotation tests equally. The eye grounds and reflexes were normal; no nystagmus; no past-pointing.

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*See page 978.

Operation, October 11, 1917. Usual curvilinear incision. On retracting the edges, the cavity was found to be occupied by a tense blue swelling. While endeavoring to obtain better exposure, it ruptured, and was surprised to get but a moderate hemorrhage, feeling that I had opened the sinus. With the cessation of the bleeding, I found that I was dealing with a hemotoma overlying the sinus, about the size of a white bean and with thick fibrous walls. The posterior portion was formed by the wall of the lateral sinus. The flaccid walls of the hematoma were trimmed. The remainder of the cavity was left undisturbed and the wound was closed. Close inspection of the posterior wall showed no apparent connection between the hematoma and the sinus. The pain which the patient had experienced was entirely relieved by the operation. Since then, however, he has at times complained of frontal headache, and occasionally a slow pulse. For this reason, he has been kept under observation, though repeated examinations of blood and spinal fluid have been negative.

Dr. Blackwell asked how long was the interval between the relief of pain and the time the patient was admitted to the hospital and operated upon for the hematoma.

Dr. Robinson replied that it was three weeks to a month.

Dr. Blackwell said that in these modified radical operations—of which he had performed some twenty-three—his experience had been that a secondary drum membrane formed across the region of the antrum internal to and over the sinus, in the area to which he understood Dr. Robinson had stated the hematoma developed. This seemed to be a primitive attempt on the part of the drum membrane to restore itself, analogous to the attempt of other tissues to restore themselves after they have been removed. All were familiar with the development of this membrane in an old radical cavity. If in any way the patient while scratching the ear or endeavoring to remove wax should thrust a pin through, one could easily understand how a hematoma might form under it—or how even an acute catarrhal condition or an extravasation of fluid might form, closely resembling a hematoma.

Case of Chronic Deafness Benefited by the Administration of Pilocarpin.

Dr. C. M. Sautter: The use of pilocarpin for the treatment of deafness has been advocated for many years, but in a perusal of the literature Dr. Sautter had not been able to find any definite data concerning the part of the auditory mechanism that is affected. The consensus of opinion seemed to be that the drug was especially efficacious in nerve deafness. He himself has used it frequently in cases of nerve deafness, otosclerosis, and in cases which he had been unable to attribute to some nasal or pharyngeal disturbance. When the pilocarpin is inflated directly into the middle ear through the eustachian catheter, the hearing and tinnitus are in some instances improved; but in comparing the tests the changes in the tone limits have always been in the lower vibrations. The question arises: Does the pilocarpin influence the change or is it induced by the routine inflation which is used to assist the injection into the tympanum, and what part of the auditory apparatus is influenced?

Occasionally he has administered the pilocarpin solution by mouth, especially in the case of patients who come from out of the city and find it difficult to return for extended treatments. In one of these cases the result was so obvious and constant that it seemed to be of sufficient importance to report.

Miss E., aged thirty years, single. First came under observation at St. Luke's Hospital in Dr. Dench's otologic service on July 6, 1915. She was employed as a bookkeeper and stated that she was afraid of losing her position because of her deafness. There was no history of any other deafness in the family. She also stated that she had been treated by a number of specialists for a considerable length of time—the treatment consisting principally of inflation—with no apparent change.

She gave a history of having had measles seven years ago, when she was seriously ill. At that time her ears discharged, and the right ear had been impaired ever since. Five years ago she fell on the back of her head and became completely deaf in the left ear. One year ago the deafness and tinnitus increased in the right ear. Both tympanic membranes were intact, but were slightly retracted and thickened. The nose and throat were apparently normal.

In testing, the left ear was totally deaf with the use of the noise apparatus, and the static labyrinth failed to react to turning or ice water irrigation. With the right ear, patient heard moderate voice at three inches, but the watch was negative. Low limit at 55 d. v.; high limit, 9,290 d. v. Bone conduction was greater than air conduction. The static labyrinth was nonreactive. These limits remained the same after air inflation. Both tubes were very patent. A Wassermann reaction was negative.

Pilocarpin solution was given to the patient, with instructions to take it to the point of physiologic reaction or until a free perspiration was induced. Upon returning the following week, she stated that she had had a most profuse perspiration, with an immediate change of hearing which was quite perceptible to herself as well as to her family. In testing her again in the same environment, the left ear remained totally deaf and both static labyrinths inactive. In the right ear, however, she was able to hear the same watch at one inch and the moderate voice at eight feet. The low limit was now 16 d. v.; high limit, 9,290 d. v.

After an interval of more than two years, the patient returned upon invitation, June 2, 1917. The comparative tests showed the hearing to be identically the same, and she stated that she had very little tinnitus.

The change in this case was in the lower vibration from 55 d. v. to 16 d. v., or normal low limit. The hearing for the voice improved from three inches to eight feet, and for the watch from zero to one inch. Since she had previously had considerable inflation with no apparent benefit, and the only treatment administered in this instance was the pilocarpin solution by mouth, it seems quite obvious that the latter induced the improvement.

Paper: Reports of Mastoid Cases From an X-Ray Standpoint.*

By George S. Dixon, M. D.

Dr. Blackwell said he did not recall the particular case to which Dr. Dixon had referred, but that he had always found Dr. Dixon's plates of the greatest assistance in diagnosis when compared with the clinical evidence. The patients also have great faith in it, and sometimes when it is difficult to get them to consent to operation they will do so when shown the X-ray plate.

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*See page 986.

Dr. Guttman said that Dr. Dixon had told of his success with these findings; and it would be very interesting if the doctor would also tell of some cases where the X-ray findings were not corroborated by the operation—in other words, if either a negative X-ray showed findings not corroborated, or the reverse. He felt sure that Dr. Dixon must have encountered some such cases. All know that almost all cases of purulent otitis media implicate the antrum; sometimes these clear up, at other times the cloudiness remains, but that does not signify an indication for operation. While X-ray findings are undoubtedly of importance, sometimes they are very misleading. It is very difficult to determine whether the condition indicates a mastoid operation.

Dr. Guttman said that while he admitted that the X-ray findings have some value, he doubted whether their value was as great as one would be induced to believe from Dr. Dixon's report.

Dr. Dixon said that he could hardly agree with Dr. Guttman in his last statement, that one can operate on a sinus on a plate alone. If there has been any disease in any sinus and it has been long continued—it does not matter how long after the affection—that sinus will be clouded. No surgeon is warranted in operating on X-ray plate evidence alone. He is warranted in operating where the X-ray plate shows that there is loss of bone if there are any clinical symptoms whatever to back it up, especially if it is increasing in size. The X-ray of any inflamed mastoid will give a cloudy plate, but that does not necessarily mean that it is an operative case.

Dr. Dixon said that very early in his work he decided not to say whether or not the routine mastoid was operative, but to report to the surgeon what was found, and to leave it to him to decide whether or not the clinical symptoms warranted him in opening the mastoid. But when he finds indications that bone has disappeared around or about the sinus, or in the squama, he feels that if there are any clinical symptoms whatever to back up the evidence, he is warranted in advising operation.

Take, for instance, the epidural abscess in Dr. Dann's case.

There was a distinct change in four days' time. We did not know it was capsulatus infection at first, but when it was found that the cavity had changed in four days' time, and that it was a capsulatus case, even though the patient had been doing fairly well, operation was advised. She had had some pain, and probably would have been allowed to drag along; but the moment positive evidence was found it was decided that it was time to operate, and Dr. Dann found the epidural abscess present at the operation.

Dr. Dixon said he did not claim never to make a mistake—he had not reached that degree of perfection—but he tried to make as few as possible. Any surgeon who would operate upon the strength of a plate alone would make a very grave mistake, unless there was some pathognomonic indication—a spot in the bone which should not be there, or that had been seen to develop during a series of X-ray examinations. Ordinarily, he must have symptoms to back it up. As Dr. Gruening used to say, "You must not forget the clinical symptoms." Blood count, blood cultivation, spinal fluid cultivation, and cell count are one point each in any laboratory diagnosis, and the plate has the same value in mastoiditis.

Paper: Clinical Observations in Subacute Mastoiditis.*

Hugh B. Blackwell, M. D.

DISCUSSION.

Dr. Seymour Oppenheimer said that there is probably no class of surgical work where the cases are so individual as in mastoid surgery. The indications for operation also are very individual; one man leans a little more to the conservative side than the other way; another leans a little more to the radical side, and the operations have to be studied by the indications in each particular case. It would almost seem as though the cases now are deviating more and more from the classical indications for operation. That thought has been brought very forcibly to my mind of late, for in spite of constantly increasing experience, one is constantly meeting with cases which seem to run such a curious type of symptoms that one is perplexed to know whether operation should be instituted or whether the case will be liable to clear up without interference. Every case seems to be a law unto itself, the determining factor often seeming to hinge on one particular symptom. In one it may be the quantity of the discharge, in another the changes in the canal wall, or in another a progressive increase in the polynuclear count.

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*See page 999.

In these subacute cases that Dr. Blackwell refers to, in one instance there may be a total lack of temperature and a total lack of the progressive fundus changes which we associate with mastoiditis; there may be an absence of all discharge; there may be a progressive polymorphonuclear count over a number of days. In another case with the same obscurity of symptoms the determining factor might be headache, or the X-ray might come to our aid and establish the indications for operative procedure.

It is difficult to understand why the cases of today seem to be different from those of ten years ago. The anatomic considerations are surely the same as ever. In individuals with a thin cortex, one would rather expect tenderness to be a prominent symptom; in a patient with a thick mastoid one would not expect any external manifestations; when there is a large pneumatic bone, one would not expect the same sharp symptoms found in mastoids constructed on the sclerotic type.

Dr. Blackwell's presentation brought up many points for discussion, but it is increasingly difficult to say what indications shall be accepted as establishing a safe and sane basis for operation.

Dr. J. M. Smith said that he thought Dr. Blackwell had not laid sufficient stress upon the capsulatus infection. In the New York Eye and Ear Infirmary he had had occasion to attend a number of such cases, and had seen very few recover without operation. The type of cases termed subacute are very apt to be those in which this infection is present. He had seen such a case in March, with a three months' history, including seven operations on the drum. When he first saw this patient the only thing to be observed was a swelling back of the ear. The drum was almost normal, showing only a slight redness along the handle of the malleus. On account of the history, capsulatus was suspected and he advised operation, and found the mastoid completely broken down. A smear showed this infection present. There was no discharge at all from the external auditory canal. Another case had had two incisions and had practically recovered, but there was a recurrence of the discharge, and operation showed a perisinus abscess with capsulatus infection.

It is especially this type of infection, after the initial symptoms of pain, tenderness and temperature, etc., have subsided, that continues to be dangerous, and so far as he had been able to determine, very few recover without operation. It would be interesting to know Dr. Dixon's experience in such cases.

Dr. Dixon said he had always taken the stand that the streptococcus capsulatus was the most insidious germ the otologist has to deal with, but that every case of capsulatus infection will do just as well as any other serious form of infection, if there is proper drainage. It is only when these cases are neglected that they become dangerous. It is the exception for a membrana tympani to become fairly normal and the mucosus capsulatus to go on with its work of destruction in the mastoid. What he called the neglected cases are those where the acute symptoms subside, leaving the patient with no pain; no temperature—nothing except discharge. When such cases continue two to six weeks, one is likely to find the mastoid a pulpy mass. That has happened over and over again in the infirmary.

However, the subacute and chronic cases of mastoiditis had given more satisfactory X-ray results than any other class of cases, for the simple reason that very frequently the X-ray would determine whether or not there was an abscess cavity to deal with. There may be few clinical symptoms and the drum membrane may look well, and yet the X-ray will show a broken down spot. The symptoms are such as uneasiness, not feeling perfectly well; a little pain one day and none the next. The patient may not sleep as well as usual. He feels that something is not quite right. Many of these cases will have existed for over a year. Dr. Coakley had sent in a case from South America. The patient was a great, strong, strapping man who came to the city on business, and went all over town to find out what was the matter with him. Some men advised operation; others advised against it. Finally Dr. Coakley decided to make a more exhaustive investigation, and had an X-ray taken. It was found that the bone was broken down below the canal and around the sinus—a rather unusual location. Dr. Atkins, who assisted at the operation, said that he had never before seen such a deep destruction in a mastoid. There was no question in that case that the deciding factor was the X-ray; otherwise, the man would not have consented to the operation.

In many of these cases the bone is almost like a piece of ivory, no cells showing anywhere. These are dangerous, for the pus cannot get out. Those who have examined many prepared temporal bones will remember that in such cases the tegmen tympani and antri may be very thin. As a rule, these circumscribed abscess cavities do not occur in large pneumatic bones; the pus makes its way out, or the entire mastoid breaks down; but with a hard bone it cannot do so, and the pus will make its way in the line of least resistance. It was in just the class of cases that Dr. Blackwell had brought up that the X-ray proved most valuable, and it was very gratifying to have him connect up the clinical symptoms with the X-ray findings.

Dr. Dixon, in closing, again emphasized the position he has long maintained, that one should not expect too much of the X-ray examination: it is only one factor, but a very important factor, in establishing a diagnosis. The plate must be backed up by the clinical symptoms.

Dr. Guttman said he was perfectly ready to admit that there is no absolute line of demarcation where operation of the mastoid begins and conservative treatment cases, and he was also ready to admit that streptococcus capsulatus cases rarely get well without operation, but Dr. Blackwell had not spoken of these cases, but of the real subacute cases found in purulent otitis media. It would be better to call such cases subacute otitis media rather than subacute mastoiditis, as we must not forget that the tympanic cavity and the mastoid antrum is only one cavity, and the affections of the tympanic cavity are the same as the affections of the antral cavity. Dr. Blackwell had spoken of opening the antrum and finding only granulations in the mastoid cells, and that the tympanic membrane sometimes showed only a small cone-shaped opening.

Dr. Guttman said he believed that if Dr. Blackwell would look into this matter further he would find that these complications are produced by insufficient drainage. In such cases, of course, sometimes the antrum must be operated; but if proper drainage is established through the tympanic membrane most of them will clear up without operation. Dr. Guttman then directed attention to the tympanic trephine which he had devised whereby a large opening could be made, enabling these cases to clear up without any mastoid operation.

Dr. Blackwell said that he had been much interested in Dr. Oppenheimer's remarks in reference to the variations of different men's individual opinions in these subacute cases, and particularly of his experience of ear surgery ten or fifteen years ago, in that it seemed to him that then there were not so many of these subacute cases. He also had made this comparison, and it seemed to him that the reason for this lies in the fact that there were not then so many specialists, even proportionally, as there are today. Many of the men then were young in years and experience, and were probably prone to operate more quickly than they are today; they did not treat the cases so long as they do now before operating. It is also probable that many of these cases were treated by the general practitioners who did not notice these subacute conditions, but dismissed them as of little or no importance. Certainly all men form their individual judgments from their experience, in seeing cases, treating them, and operating upon them; and if, unfortunately the patients die, by postmortem examinations.

So far as the capsulatus infection is concerned, Dr. Blackwell said that he had stated in the paper that he considered all the streptococcus infections to be the most serious ones, including the capsulatus.

Replying to Dr. Guttman, he said he was sorry the doctor received the impression that he was speaking of otitis media rather than mastoid conditions; he had discussed subacute mastoid conditions. The semiresolution in these cases is caused by the fact that the middle ear cannot communicate with the mastoid. It is absolutely plugged. The middle ear not being reinfected from the mastoid, quickly undergoes resolution, and if anyone bases his opinion on the appearance of the middle ear he is apt to have a sad awaken-ing. Dr. Blackwell said that he had had one very bitter experience in this respect and hoped not to have such another. One can speak of resolution as applied to the middle ear, where it is possible to actually observe it, but not as applied to the mastoid. The condition there may go on to almost anything—subperiosteal abscess, brain abscess, etc.

NEW YORK ACADEMY OF MEDICINE. SECTION ON OTOLOGY.

Meeting of December 14, 1917.

Acute Serous Labyrinthitis Complicating Acute Suppurative Mastoiditis.

DR. JOHN McCOY: Giovanni La C., aged fifty-five years, Italian, was admitted to the hospital November 15, 1917, with the following history:

About the 5th of November, patient had an attack of the grippe which affected the nose and nasopharynx. Ten days later the patient noticed a pain in the left ear, which became quite severe. Four days ago a discharge started from the left ear, which has been copious in amount and of offensive odor. Patient stated that he had had severe vertigo since the trouble with his ear started. He said he felt as though he was drunk and could hardly walk, and that he had a tendency to fall to his left. The patient's relatives stated that he had never had any trouble of this kind previously.

Examination of right ear negative. Examination of the left ear showed the canal filled with purulent discharge, after the removal of which the membrana tympani seemed to be perforated, red and bulging above. No tenderness over the antrum; very slight over the mastoid tip. Temperature at time of admission, 100° F.

Functional examination showed hearing for the watch in the left ear negative; the whisper, fourteen inches; the voice, sixteen feet. Weber test referred to the right ear. Spontaneous nystagmus to the right. Turning test showed the right labyrinth functioning normally. Patient standing with eyes closed, fell to the left. Examination of the smear showed staphylococci. X-ray plate showed fairly pneumatic mastoid, both sides; the left quite cloudy. Examination of spinal fluid showed it to be clear and cells fifty-five per millimeter. Globulins plus. Fehlings plus. Cultivation negative.

Operation, November 17. On opening the cortex, pus and granulations were removed and the mastoid bone was found to be extensively involved. The sinus was exposed for about one inch and epidural abscess found; the mastoid antrum was filled with granulations and pus. The patient was returned to the ward in good condition, and for the next three days his temperature ranged between 99 and 100.5 degrees. On the third day a complete dressing of the wound was done, which was found healthy. The patient's general condition was very much better. His dizziness, he said, was a great deal less, and the nystagmus was practically absent.

On November 25th the nystagmus had entirely disappeared. On turning to the right, he had nystagmus to the left for six seconds. With the noise apparatus in his right ear, he was unable to hear in his left ear the loudest voice or the Galton whistle. Weber was localized to the right side. The patient continued to improve, and he was discharged from the hospital on December 7th. He still returns for dressings. His labyrinth reactions are still about the same.

DISCUSSION.

Dr. Alfred Kahn said that he believed that acute labyrinthitis followed acute otitis media more frequently than was generally appreciated. Recently he had seen two cases in which dizziness developed after an acute process in the middle ear. This dizziness was associated with nystagmus toward the opposite side. In one instance the dizziness and nystagmus lasted over a period of two or three weeks, and the hearing in the diseased ear was entirely lost. In the other case which he has still under observation the nystagmus stopped after a period of a week, but the patient still feels dizzy. She is evidently getting well—her symptoms abating. Her hearing is slightly reduced in the diseased ear.

In such cases the prognosis as to hearing should be carefully remarked upon. Dr. Kahn said he had found that tincture of aconite gave some relief in acute processes of the labyrinth. He started with five drops, the patient being instructed to increase the dose one drop each time the medicine was taken. The drops were given three times a day. Some patients had been able to increase the dosage even up to a teaspoonful without any ill effect.

Pathologic Exenteration of the Middle Ear Without Operation.

Dr. Frank H. Knight (by invitation): The patient was a longshoreman, forty-eight years of age, born in Roumania. He was referred to Dr. Knight by Dr. Ittelson for treatment, and two days later was admitted to the Manhattan Eye, Ear and Throat Hospital in Dr. Duel's service. His chief complaint was an itching sensation in the left ear and dizziness. This dizziness was at times so severe that he staggered and gave the impression that he had been drinking, which he claimed he did not do. Dr. Ittelson removed a mass of cerumen, epithelial cells, etc., from the ear.

Dr. Knight said that when he examined the patient there were no signs of any operative interference, either within or outside of the ear, but the external auditory meatus was larger than normal, and about one-half inch internally the canal suddenly enlarged into a large cavity. The posterior limits of this cavity could not be seen, but it seemed to go back into the region of the mastoid cells. The drum membrane and ossicles had disappeared; the posterior wall had been lowered, the canal and mastoid cells widened, so that the tympanic cavity, aditus, antrum and mastoid cells formed one large cavity.

It was an interesting question as to how this pathologic exenteration had taken place. Dr. Knight said he had recently operated on three cases in children, two at the Manhattan Eye and Ear Hospital and one referred by Dr. Page of White Plains, New York, in each of which there was a subperiosteal abscess, and on making the usual incision, retracting the soft parts and wiping away the pus and granulations, a considerable portion of the posterior wall of the external auditory canal was found to have become softened and disappeared. In one instance the lowering of the posterior wall was almost complete.

Dr. Knight said he thought that in this case there was probably a similar subperiosteal abscess. The wall became softened and disintegrated, and the abscess broke through into the canal, and that during the period of years following (thirty-two, he understood), the suppurative process had continued, the exenteration had taken place, resulting in the configuration as seen in the present large dry cavity.

After he had seen the patient two or three times, the man disappeared from observation, and he had not been able to make the ear tests as he would have liked. The man can hear the whispered voice eight inches or a foot away.

The dizziness was an interesting point, for it came on when the man was doing hard labor, when he puts his head back; or if he shuts his eyes in a street car he becomes dizzy. It has now lasted for twelve years. He has an ocular nystagmus but no spontaneous nystagmus.

An X-ray plate had been taken by Dr. Frederick M. Low, which showed very clearly the difference between the two sides. The right side is apparently normal, showing clearly the outlines of the cells; the left side shows the external auditory meatus much larger than normal. No cells are to be seen, but posterior to the sinus there is a sclerosis.

Dr. Knight said he believed the condition to be an uncommon occurrence.

Dr. Guntzer asked if there were any caloric symptoms. Did the man feel especially dizzy this cold weather?

Dr. Knight replied that the man did not complain particularly of cold air, though these cases usually do. The dizziness came on when walking, or when he lies down or throws his head back.

Dr. Hurd asked how Dr. Knight accounted for the fact that the labyrinthine process came on twenty years ago, and the history that the man who referred the patient got out a lot of waxy material. In his opinion it looked more like a cholesteatomatous exenteration, and there might be some cholesteatomatous process still going on.

Dr. Guntzer said he did not think it a fair inference that the pathologic process was the same in this case and in the child's case which Dr. Knight cited. The man had had this process for thirty years. Was there any odor?

Dr. Knight replied that there was no odor, and that he found no evidence of any discharge.

Dr. Forbes asked if the absence of odor would not rather negative the theory of a cholesteatomatous process.

Dr. Guntzer said that it was a very interesting case. The ear seemed perfectly smooth and perfectly dry. Such cases were not very common.

DR. KNIGHT, replying to Dr. Hurd's comment, said that he had tried the fistula test, but had not been able to detect any fistula. He could not say why the dizziness should come on after ten or twelve years. On account of not being able to keep in touch with the patient he had not been able to make as thorough a test as he would like. The Wassermann test had not been made, nor examination for evidence of tabes. In regard to cholesteatoma, very likely there had been formation of cholesteatoma, for there seemed to be epithelial cells in the mass Dr. Ittelson removed. Dr. Amberg, in 1912, mentioned a similar case, the only one he (Dr. Knight) had been able to find.

Having seen the other cases recently, he was inclined to believe that this one also might have occurred in that way, and to feel that it was due to a subperiosteal abscess breaking through, as the starting point.

Extensive Tuberculosis of Both Mastoids and Middle Ear.

DR. L. M. HURD: The patient was a little girl, seven years of age, who came under his observation early last spring with a history of mastoid trouble in both ears, of three months' duration. The ears were still running, and there was a fistula behind one ear. There was some swelling over the left mastoid and considerable induration of the glands of the neck on both sides. The condition aroused suspicion of tuberculosis, and she was sent to the hospital. The mastoids were opened up and were found to be entirely filled with a gelatinous granulation tissue and a kind of flocculent fluid. Feeling convinced that it was a case of tuberculosis, Dr. Hurd did not clean out the mastoid. The condition extended from the semicircular canals into the petrous portion. This also he did not clean out, but closed the wounds and treated the case with bismuth paste. Three months later he saw the child, and one ear was well; the other had a sinus running back an inch and a half. He instructed the parents to resume the bismuth paste treatment, and recently he had had a letter saying that the child was in fine condition. The hearing test was five inches right and one-half inch left with the watch. The patient was one of those crying children, and nothing could be done with the labyrinthine test. Apparently, however, the labyrinth was intact within the granulating mass.

Dr. Hurd, replying to an inquiry from Dr. Guntzer, said that the pathologist reported the condition to be a tuberculous granuloma. In his own opinion, the less one does for a tuberculous mastoid, the better for the patient.

Acute Mastoiditis Complicated by Thrombosis of the Sigmoid Sinus Extending Down Into the Jugular Bulb and Internal Jugular Vein.

Dr. L. M. Hurd: The patient was a woman apparently about thirty-five years of age. Two years ago Dr. Hurd was called up by another physician, who asked if he could do a mastoid operation that day (Thanksgiving). He said that the patient was an employee in a hotel who had been missed from her usual position, and upon inquiry was reported to be ill. Later on, one of the attendants said that she was being treated by Christian Science for ear trouble, and was growing worse. Then this physician went to see her and found her in a very pitiable condition, with an enormous mastoid swelling.

Dr. Hurd agreed to operate that afternoon, and she was sent to the hospital. On reaching there about four o'clock, the patient was put upon the operating table, and he opened up this mass. Under the periosteum was about two ounces of pus. Upon evacuating this fetid pus the cortex was found to be purplish and congested, but there was no perforation. Evidently it was a Bezold. The interior of the mastoid was a mass of soft mushy granulations. The dura was exposed over the antrum and sinus, and in the vein a thrombosis was found. A free flow was obtained above the knee, also below the thrombosis. The pus had burrowed between the dura and the skull, between the skull and periosteum. An extensive piece of bone was removed, and the tip and petrous cells were removed nearly to the foramen magnum. The pus had burrowed down the muscles of the neck across the transverse process and along anterior to the sternomastoid muscle. This was evacuated. The patient was in very poor condition. Before the operation she was asked if she had had any chills, and she replied in the negative. She had the attitude of the Christian Scientist, that nothing was the matter with her.

That night she had a chill, and a temperature of 106. The next day she went on all right, but the next day she had another chill and high temperature. On Saturday she had another chill while waiting to be sent to the operating room. On reaching there she was nearly moribund. The neck was opened without anesthesia. After this the patient was returned to bed and made a prompt and uneventful recovery.

The pus pockets went down in three or four directions. No counter drainage was instituted, but the pockets were filled with bismuth paste. A general surgeon who was present said counter drainage of the pockets only would cure, to which Dr. Hurd replied, "All right," and went on injecting bismuth paste. In about three weeks after the operation all the sinuses down to the periosteum and bone had healed and the wound was entirely dry in January.

Dr. Hurd said he had not seen the patient for about a year, but had asked her to come to the meeting tonight.

Dr. Voislawsky said that Dr. Hurd's case was a demonstration of the fact that it is a great mistake not to ligate the jugular in a case of sinus thrombosis. He disclaimed any idea of criticism of Dr. Hurd, for he would certainly have done it if the condition of the patient had warranted it. Dr. Hurd was certainly to be congratulated on the successful issue of the case and in being able to present the patient in such excellent condition. The case merely brings up again the question that has been thrashed out so many times—about excising or ligating the jugular, when the sinus contains a clot or is thrombosed. In this case the jugular was not excised, only tied off. It is quickly and easily done, and the physician and the patient would both be saved much if every thrombosed sinus was so treated.

Fistula of External Semicircular Canal Occurring in a Case of Acute Suppurative Mastoiditis.

Dr. John McCoy: J. M., aged sixty-four years, a native of Canada, entered the hospital August 8, 1917, with the following history:

Following a cold in the head, he developed pain and fullness in the right ear. The right drum was opened on July 6th. The ear discharged for two weeks and then dried up. Since the discharge stopped, he had had a dull pain over the mastoid and over the right side of the head. When examined on August 8th, which was about five weeks after the beginning of the attack and about three weeks after the ear discharge had dried up, he presented the following conditions: The external ear was dry, the ear drum appeared congested and somewhat thickened, and without any balging. There was a tenderness over the mastoid antrum and the tip, elicited on pressure. His temperature was normal. An X-ray of the mastoid showed an abscess in the mastoid cavity with a melting away of the bony partitions. A mastoid operation was advised and performed the following day.

The operation disclosed the following conditions: Granulations and free pus throughout the entire mastoid. The inner plate over the sinus was eroded from practically the knee to the bulb and there were granulations and pus on the dura beneath the lower part of the sinus where it swings anteriorly into the bulb. There was also a well marked fistulous opening into the external semicircular canal. As the patient had complained of no symptoms referable to his labyrinth, it was decided not to operate on the labyrinth. The mastoid was thoroughly exenterated and the patient was returned to bed in good condition. The day following the operation his temperature was 100° per rectum, and his convalescence was apparently normal in every way for five days. During this time the patient said he felt perfectly well; that he was not now nor had he been dizzy or unsteady in his gait. On the fifth day his temperature rose to 105°, and he was again taken to the operating room. A spinal puncture was made and turbid cerebrospinal fluid was withdrawn. The wound was reopened along its entire length and found to be healthy. The sinus was opened to see if there might be a suppurating clot within, but it was found to be healthy and bled freely from both ends. The cerebrospinal fluid showed streptococcus in long chains. The patient failed very rapidly and died the following day.

This case was presented as a companion to the preceding case. In the one, apparently serious labyrinthine symptoms subsided with conservative treatment. In the latter, there is a possibility that the meninges became infected by way of the fistula in the external semicircular canal, and yet the writer did not feel that such was the case, without giving any symptoms of the labyrinthine irritation, but rather that, due to free pus in contact with the dura and no drainage for several weeks, the infection spread through the dura.

Pneumonia With Interesting Otitic Complications.

DR. SEYMOUR OPPENHEIMER: B. S., aged fourteen months. Seen in consultation on October 31st. Child became ill about October 10th, with a temperature averaging about 102° for a period of eighteen days. Diagnosis, influenza. On October 28th the temperature rose to 104°, and Dr. E. Riesenfeld assumed charge of the case.

Examination showed a marked state of congestion of the mucous membranes of the upper respiratory tract. After two days' observation, the temperature ranging from 104 to nearly 106 degrees, a questionable area of consolidation, very small in size, was discovered at the right posterior lobe of the lung. The drum membranes were markedly reddened and bulging, and a double myringotomy was performed by Dr. Milton Ballin. As the discharge was very scant and the temperature did not subside, and as the tympanic membranes showed the evidence of a lack of free drainage, the following day myringotomy was again performed, followed by a profuse purulent discharge from both ears. A blood culture taken at this time was positive, the pneumococcus being found. No ear culture had been taken. On October 30th the temperature after a remission from 105 to 99 degrees, rose sharply to 107°, followed by a rapid fall to 98°, and then rose again sharply to nearly 106°. At this time I saw the patient, and in analyzing all the facts determined, firstly, that there was no demonstrable disease of the mastoid, and concluded, in view of the fact that no absolute assurance could be given me that a pulmonic process was present, that we might be dealing with a primary jugular bulbar thrombosis. I expressed the opinion, however, that in a series of over one hundred cases of sinus thrombosis I had never found a case where a pneumococcus was present in the blood stream, the organism being always the streptococcus or streptococcus mucosus, and that I would advise deferring interference a short period to determine whether some more definite lesion could not be found in the lungs.

I might say in passing that it is not infrequent to find the pneumococcus in the blood in the early stages of a pulmonic process. A day or two later the local ear symptoms had not progressed, and the attending physician definitely stated that there were the evidences of a right lobe consolidation. The temperature remained at 105 to 106 degrees for several days and subsided by lysis with a complete recovery of the patient.

This case is simply presented as showing the possibilities of an error in diagnosis.

Extradural Abscess and Fulminating Meningitis.

DR. SEYMOUR OPPENHEIMER: A. L. H., aged eight years. For two days patient had a slight cold with a mild temperature, and complained of occasional pain in the left ear. On the evening of June 12th the patient was seen by me at Elberon, New Jersey. After examination a myringotomy was performed, which procedure was followed by a few drops of serum from the middle ear cavity. Slight pain was complained of upon pressure over the antetragal region. The myringotomy was followed by a relief of the pain, and the temperature dropped to normal. The child fell asleep shortly thereafter.

Later in the evening the little patient became very restless but did not give the parents any anxiety until midnight when some delirium manifested itself. At this time Dr. Edwin Steirberger and myself were hastily summoned. We found the child in absolute coma, with marked convulsive movements of the entire right half of the body, the face being paralyzed. The left pupil was dilated and the right retracted. Opisthotonos was extreme. An external strabismus was noted and nystagmoid movements of the eyes were pronounced. Temperature 105°, pulse 180, respirations 36. The patient was promptly placed in a hot bath and morphin administered, and arrangements were quickly made to remove the child to the Monmouth Memorial Hospital at Long Branch, several miles distant.

The mastoid process was quickly opened and found normal. A large area of bone was removed over the squamous plate of the temporal bone. The dura was found to be extremely tense, but did not show at this site any inflammatory process. Suboccipital decompression was also performed by removing an area of bone posterior to the mastoid.

In searching over the extradural spaces, a few drops of pus were located well anterior over the root of the zygoma. A large portion of bone was removed in this direction so as to expose this region thoroughly to view. A mild inflammatory process of the dura was noted at this point. A lumbar puncture was then made, and about ten cubic centimeters of fluid under considerable pressure and slightly turbid in character was removed. No microorganisms were found in the smear nor were any found in the cultured material, but cellular count was markedly increased.

After all the operative procedures had been completed, Dr. Charles Elsberg arrived from New York, and we remained that day in constant attendance upon the child. The temperature had dropped steadily to 99½°, and the comatose state cleared up, the entire picture improving following the cranial decompression means employed. Other than the complaint of headache no symptoms were manifest, although a definite bilateral Kernig was recognized, with some neck rigidity.

The following day, June 14th, some retinal congestion was noted and a definite optic aphasia, which, as well as the Kernig and neck rigidity, decidedly improved for the ensuing four days. The patient apparently was much improved, although the temperature showed a steady tendency to rise each day a trifle, reaching to nearly 103° on the afternoon of the sixth day. At this time the dressings were removed and the wounds were found to be very clean. The evidences of a meningeal irritation became more manifest, and at Dr. Elsberg's suggestion Dakin's method of flushing was employed over the exposed dural areas; on the following day lumbar puncture was again employed, and fifteen cubic centimeters of fluid under great pressure was removed. This contained some seventy per cent of polynuclear cells, but again no microorganisms were found in the smear.

The brain was exposed in various directions by Dr. Elsberg and myself, but no localized abscess could be encountered. The dura over the tegmen tympani was then incised and a large area of brain tissue was found to be softened and breaking down. This encephalitic process accounted well for the symptoms of aphasia. From then on all evidences of a rapidly spreading meningitis asserted themselves, the patient succumbing the following day.

Only in the last lumbar puncture fluid were microorganisms found, they being the streptococcus mucosus. The interesting features of this tragic case were the violent onset of the symptoms, the relief of the same after the decompression operation, the absence of all the disease in the mastoid, the route of infection being anterior through the zygomatic root, the finding of an extra dural collection of pus at this site, starting from which an area of encephalitis developed, which of course was beyond the reach of any surgical intervention.

Sinus Thrombosis With Metastases.

DR. SEYMOUR OPPENHEIMER: J. W., aged ten years. Admitted to Mt. Sinai Hospital July 30, 1917.

Previous History.—Since ten days, purulent discharge from right ear with some temperature.

Examination of the ear showed a moderate amount of secretion in the external auditory canal of the right ear, with a small perforation in the anterior inferior quadrant. No evidence of retention, nor any mastoid involvement.

The case was kept under observation in the ward of the hospital. Six days later some slight neck stiffness was noted with a suggestive Kernig and Babinski. Also a beginning papillitis. The ear was apparently resolving. Lumbar puncture showed an increased cell count to 36 per cubic millimeter, with a polynuclear count to 66 per cent. Temperature rather stationary, about 102. Blood culture and widals negative.

On the eleventh day after admission the neck rigidity, Kernig and ankle clonus becoming more marked, but the ear symptoms abating, pain was complained of in the left shoulder and right ankle. Blood culture taken at this time was reported as showing the streptococcus hemolyticus. White blood count, 24,000. Polynuclear count, 85 per cent.

At the time of the submission of the above report, tenderness and swelling appeared over the right and left knee, and two chills were experienced by the patient, followed by a considerable rise of temperature.

An operation was advised upon the mastoid and sigmoid sinus, as it was reasoned that these articular swellings were the metastatic evidences of a thrombotic process. Exploration of these regions failed to show any involvement of the mastoid, nor could any thrombus, even parietal, be demonstrated, although at one point the vein wall appeared rather thickened. The jugular vein was not tied at this time, but was subsequently ligated, as the blood cultures remained positive, which was followed several days later by the report of a negative culture. During this period a large number of metastatic conditions manifested themselves, such as effusions into various joints and abscesses of the skin. The left hip joint required surgical interference, and a large quantity of pus was evacuated from this site. A general septicemic process developed, from which the patient succumbed on September 20, 1917.

Among the numerous interesting points which this case brings up for thought is, firstly, whether in the presence of the evidences of an arthritic process and a coexisting aural suppuration and a positive blood culture, whether it would not have been a justifiable plan to have immediately ligated the jugular vein, even though a demonstrable thrombosis could not be determined, and again, in the presence of the above mentioned groups of symptoms, namely, a metastatic process, a preexisting aural suppuration and a positive blood culture, whether this could not be construed as the definite evidence of a phlebitic infection.

Mastoiditis With Meningitis Arising Four Weeks After Operation.

DR. SEYMOUR OPPENHEIMER: M. T., aged forty years. Admitted to my service at Mt. Sinai Hospital on July 7, 1917, complaining of pain over the left side of the head since four weeks, during which time there was some temperature and a loss of weight of fifty pounds. The previous day a spontaneous bloody discharge took place from the left ear.

Examination showed some tenderness over the mastoid tip and posterior border, with a profuse purulent discharge from the external auditory canal, with some evidences of retention. Myringotomy was promptly performed.

Hearing for conversation voice with exclusion of the unaffected ear was six feet, with the Weber lateralized to the affected side. The static labyrinth was demonstrated to be functionating normally. Ocular examination negative.

Two days after admission a mastoid operation was performed, at my direction, by Dr. Auerbach. The operative findings consisted of a perisinus abscess with an epidural collection of pus over the tegmen, in the middle cranial fossa. Bacteriologic examination of this pus showed the presence of the streptococcus mucosus. Four weeks after the operation, the patient convalescing in the hospital with the mastoid wound almost closed, the temperature rose suddenly to 104°, with the rapid development of all the signs of a meningitis. Lumbar puncture was immediately performed. The spinal fluid was under great tension and very turbid, and contained 260 cells to the cubic millimeter, with a 98 per cent polynuclear count. No organisms were found in the smear, but very abundant in the culture (the streptococcus hemolyticus). An exploratory craniotomy and temporal decompression was promptly made, but no route of infection could be determined.

Some hours later the testing of the labyrinth showed the same to be absolutely nonfunctionating. Fundi negative. The general condition of the patient being so critical, an operation upon the labyrinth was not undertaken. Death ensued in thirty-six hours after the onset of these symptoms. An autopsy could not be obtained.

The interesting features which this case brings up for discussion are: Firstly, the unknown possibilities of reinfection, or rather the lurking of infection in these cases of streptococcus mucosus; and secondly, how without any recognizable symptoms a labyrinth which was demonstrated to be functionating normally became the seat of a probable suppurative process, with the ensuing meningitis, while the patient was up and about in the hospital under proper observation, proving how rapid may be the transition from an unrecognized perilabyrinthial inflammation to a complete destructive process.

DISCUSSION.

Dr. Carter asked if Dr. Oppenheimer did not think that complications involving the mastoid and the sinus were fairly uncommon in infections of the middle ear accompanying pneumonia. Last year in the Children's Wards of Gouverneur Hospital there were a great many pneumonia cases—over a hundred—and fully half of these cases had involvement of the middle ear, yet not a single one of those cases was complicated by mastoiditis or sinus involvement or other complication of middle ear trouble.

He also asked if Dr. Oppenheimer had not noticed in the epidemic of pneumonia last year an unusual proportion of cases with middle ear conditions. This year also there has been quite a number of cases of pneumonia in the children's wards, but so far he had not seen any middle ear involvements; whereas, last year they quite got into the habit of expecting it.

DR. GUNTZER said it was commendable that Dr. Oppenheimer reported these cases from the negative point of view, as we usually get the other side. He had seen a parallel case in the country, but having no facilities for blood culture he had to rely entirely on the physical and clinical symptoms, for the physician in charge would not send the case to the hospital. There was no clinical evidence of any kind of mastoid involvement; there was the jumping septic temperature, but on the fourth day he made out a pneumatic process. We ought to keep in mind the clinical symptoms end of it, along with the laboratory findings.

DR. HURD said he thought there was liability of the ear condition overshadowing the general condition. Recently an Italian was brought into the hospital with a history of having had the mastoid operated on the week before, and since he had been running a very high temperature and had developed a facial paralysis. When seen in the hospital his temperature was 105° and he had a dead labyrinth and a little bronchitis, which was thought due to the anesthetic. However, it was decided to go in and do what could be done. At the operation an enormous cholesteatomatous mass was found invading the labyrinth; the sinus was opened but nothing found. The facial nerve was affected by the pressure. The patient was returned to bed and developed a temperature of 105 to 106 degrees. A physician was then called in, and he said the patient was developing an acute miliary tuberculosis of the lungs, and the man died of tuberculosis. While he needed to have something done for his ear, his serious symptoms had nothing to do with that condition.

DR. KAHN said he judged from Dr. Oppenheimer's report of his case of meningitis that the patient progressed fairly well up to the time that the dura was incised. He thought it was poor surgery to incise the dura for the relief of brain tension in the presence of an infection, this notwithstanding the fact that the procedure was considered correct. He thought that it was all right to expose a large area of dura for decompression purposes, but he could not see the rationale of exposing the subdural space to infection. In looking over the literature on this subject he had noted a very large percentage of deaths where the subdural space was opened. In the labyrinth operation (Richards) operators were extremely careful not to expose the subdural space. Why should not this law hold for the subdural space in other localities? He did not think the results warranted the procedure.

Dr. Hurd, referring to the case of mastoiditis with meningitis, said that a year ago he was called to a town in New Jersey in see an eleven months old baby who had had a mastoid operation performed four weeks previously. (A week after the operation he had had a flush of blood at time of dressing.) The mastoid wound had practically healed leaving only a small scab, and the middle ear was dry. For three or four days the child had been running a high intermittent temperature, ranging from normal to 104-105 degrees. The surgeon who performed the mastoid said that the mastoid condition was all right, but the family wanted Dr. Hurd's opinion. Dr. Hurd said he knew the family physician, who was good with children and had confidence in his judgment. It was thought that the sinus might be the cause of the trouble. Accordingly it was decided to operate, and a small sigmoid sinus was uncovered, but there was no thrombosis or thickened wall. It was opened up, however, and the jugular vein was tied off. The child had no further rise of temperature and got well. He had not seen it since.

Dr. Seymour Oppenheimer, replying to Dr. Carter's inquiry in regard to middle ear infections in pneumonia, said that they were not so frequent as are seen in many other acute infectious diseases, though many cases do have red drums requiring myringotomy. That, however, was not the point he had tried to make. He was trying to emphasize the confusing thought that might arise on finding pneumococcus in the blood stream, presuming that the clinical picture would be suggestive of a sinus thrombotic process; that his experience to date has been that the pneumococcus has never been recovered from the blood in any of his cases of sinus thrombosis. That is important in many ways from the prognostic standpoint. For example, a mastoid operation has been performed and there has been recovered the pneumococcus from the mastoid pus; subsequently the patient begins to run a temperature that might be considered as due to a sinus thrombosis. He has felt reasonably safe in saying that the patient was not developing a sinus thrombosis if the mastoid pus had demonstrated the pneumococcus. This opinion has always been justified subsequently by finding some other explanation for the symptoms other than the existence of a sinus thrombosis.

Dr. Carter had asked if last year there had not been more ear complications in the pneumonia cases than in other epidemics. Dr. Oppenheimer said that this had been observed, but that the explanation lay in the fact that many of the pneumonia cases had been complicated with influenza, in other words, a mixed infection.

In regard to the case of extradural abscess, the question had been raised as to the wisdom of the dural incision. Dr. Oppenheimer said he was thoroughly in accord with the statement that the dural incision is very bad practice; but in this unfortunate case the dura was not incised for the purpose of draining the subdural space. Some days later, however, when the severe symptoms indicated that the patient was in a very critical state, the brain was punctured in various directions in the hope of finding the abscess, and it was then that a dural flap was made and a large area of encephalitis was found. Dr. Oppenheimer said he thought subdural drainage was a delusion and a snare; what cannot be accomplished by lumbar puncture cannot be accomplished by subdural drainage. With the exception of cisterna magna drainage, at any other site you have within a very few hours a pretty definite binding together of the pia arachnoid and dura, and drainage does not take place.

Dr. Oppenheimer said he was quite in accord with Dr. Voislawsky in feeling that one is exposing the brain to added infection by this practice, and he was satisfied that in every case of meningitis where the dura has been incised, that probably an additional factor has been supplied which has hastened the end.

Paper: Notes on a Case of Meningitis of Otitic Origin.*

By Alfred Kahn, M. D.

DISCUSSION.

Dr. Hurd asked what Dr. Kahn meant by the expression "cerebral decompression." It was a little confusing.

Dr. Kahn said that cerebral decompression is very often done as a therapeutic measure in meningitis. His idea is that it is practiced in two ways: First, by removing the bony cortex of the skull and exposing a large area of dura, the idea being that the brain will then have room for expansion, as the dura offers less resistance than the bony skull; second, cerebral decompression is practiced by going a step further and incising the dura, exposing the subdural space. This procedure not only allows for expansion of the brain substance but is supposed to drain the subdural space, thereby relieving tension. The danger of this second procedure is that, should infection enter the subdural space the result is almost sure to be fatal, as once the brain is invaded beyond the dura—the dura is the strong line of resistance between the brain and the outside—serious results are almost sure to follow.

Dr. Hurd responded that the dura is a pretty tough membrane and does not bulge much. When you really want to decompress the brain you have to go into the temporal region and open the dura.

Dr. Kahn replied that the dura is not as resistant as the bony cortex.

Dr. Kahn stated that the method is practiced. As to its value he is not in a position to say. It would seem, however, that the dura offers less resistance than the bone, and that where an area of bone is removed the dura will tighten (hernia) into the opening, thus reducing tension. Of course the decompression is not as effective as where the dura is incised, but the latter procedure markedly increases the risk. He is not in a position to state what decompression value merely exposing the dura over a large surface has, but he does know that the method is practiced.

Dr. Hurd said that was another question. Can you have a hernia with dura intact?

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*See page 1004.

Dr. Guntzer asked if Dr. Kahn had any idea where the pressure was relieved.

Dr. Kahn stated that the case was one of meningitis. The patient was unconscious and delirious. He had an extremely high temperature. He had stiffening of the neck and back. Kernig sign was present. He had a high blood count. He had an unusually high spinal fluid cell count. He unquestionably had meningitis.

The Second Operation.—The patient was taken to the operating room the second time merely to examine the wound from the previous operation to see if there was any discernible reason for his symptoms—as a routine matter, in order to give the man any possible chance that such an examination might offer. The dressing was removed and the wound examined, but nothing was found. On account of the high temperature the lateral sinus was again opened and explored between the mastoid opening and the torcula, but no point of infection was located. The sinus bled very freely from above. This was the only thing accomplished by the second operation. The temperature promptly dropped and the next morning the patient regained consciousness and later recovered.

Dr. Guntzer said he had asked the question because some years ago Dr. Haskin presented a good many anatomic studies in discussing the cause of abducens paralysis and papillitis; and with the aid of these studies he worked out a definite reason for it—a vascular or sinus pressure at the foramen.

Dr. Voislawsky asked if it was not possible that the bleeding might have washed the clot away, inasmuch as the clot is not always apparent in the opening.

Dr. Kahn replied that it was possible that it might have happened, but he did not think that had occurred, as the blood was very clear.

Dr. Oppenheimer said he did not think one could call it a decompression operation when only a small piece of bone was removed. In the decompression operation of the general surgeon a large osteoplastic flap is removed. The benefit he believed to be as much due to the lumbar puncture therapy as to the removal of a small area of bone. One should not be homeopathic in taking out a flap of bone.

He could not see the rationale of the sinus bleeding which

Dr. Kahn referred to as a decompressive measure, for you replace the same amount of pressure from the packing necessary to control the bleeding. It is a question whether that does not exert more pressure on the brain than the amount of blood lost.

He did not believe that one ever gets a hernia from a small exposure of the dura. You do get a hernia when you incise the brain—and the brain is infected. Cerebral hernia is a sign of brain infection, excluding that type of case, where you have removed the entire roof of the mastoid, the wound has healed and some reinfection takes place subsequently, and increased intracranial pressure, and there is prolapse of the brain with the dural covering into the exenterated cavity; but from simple exposure of the dura he doubted whether a hernia would result.

Dr. Kahn further stated that he had not had any very great experience with such cases, and that he merely reported the case stating what he had done and giving his own ideas about the matter. He simply stated his ideas for what they were worth, not as a fact. As previously stated, the only thing accomplished at the second operation was the free bleeding from the sinus. The patient's prognosis was practically hopeless, and in the absence of anything else other than the free bleeding, he assumed that recovery was due to this fact. Regarding brain hernia, he did not want it understood that you could have a hernia in the sense that a large portion of the organ could push itself through the bony cortical opening. The brain has a tendency to tighten itself into the opening, but as a matter of fact the operation of exposing a large area of brain without incising the dura is often done. If it is not done for decompression, he would like to know what is the purpose.

Dr. Oppenheimer, referring to the spinal fluid findings, said that it had never come within his experience that the cell count had been as high as in the case reported by Dr. Kahn, and bacteriologically sterile. He knew of many instances where the cell count had been high and no bacteria found in the smear, but they had always been found in the cultures. This was a case of an increased leucocytosis of the spinal fluid, and might be construed as aseptic (serous) meningitis rather than a bacterial meningitis. Otherwise he was satisfied the patient would not have recovered.

Dr. Kahn further stated that the spinal fluid contained cells equal to pus; the culture was sterile. Fehlings was minus, and the globulins were four plus the morning of the second operation. The globulins were later reduced to two plus on the next spinal fluid examination after the operation. This is mentioned to show that although the fluid was negative (no germs being found) there must have been some kind of an infection to cause a minus Fehling and such a high globulin reaction. It might be that the spinal fluid has some germicidal effect.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of November 20, 1917.

Dentigerous Cyst.

Dr. Joseph Beck presented a patient with a dentigerous cyst. The patient was a little boy who had a unilateral swelling of the right side of the face, which was hard on palpation and which had been present for about ten months. There was associated with it a very foul odor from the mouth. It was not painful. On palpation directly over the swelling in the mouth an indentation could be made which was effaced when the pressure was removed. It felt like pasteboard, did not give a crackling sound, but responded to pressure. This was the cardinal symptom of a dentigerous cyst, which was the diagnosis. The diagnosis was proved by X-ray pictures taken both in films and in posteroanterior exposure. The teeth in the cyst were merely formed and unable to erupt. They appeared to be the incisors and premolars. Puncture of such a cyst usually revealed fluid of a gelatinous nature. Dr. Beck showed pictures of other dentigerous cysts.

Dentigerous Cyst.

Dr. Harry L. Pollock showed a young man whom he had seen for the first time on the previous morning. He complained of a swelling over the right antral region, which had been present for about a month. During this time he noticed that the right side of the nose was not as free as the other. There was no pain, but when he bit on that side he could feel the teeth give a little. A very soft tumor, which was crepitant, could be felt over the antrum. A dentigerous cyst was suspected and the X-ray examination proved that diagnosis correct.

Vibratory Mechanism.

Dr. Robert Sonnenschein demonstrated a small vibratory mechanism which could be used as a noise apparatus in testing for unilateral deafness. It was operated by a small, dry cell battery which retailed for about thirty-five cents. The appliance was made by Sharp & Smith, and sold for $5.00. The other noise apparatuses previously used were the Neumann, which was very expensive, and the Bárány, which is now practically unobtainable on account of the war, and it had cost a considerable sum.

The disadvantage of this instrument was the fact that a good deal of outside noise was being produced, but efforts were being made to overcome this defect. In testing patients, however, with the apparatus in one ear it was found that despite this noise they were able to hear even the whispered voice in the other ear.

The advantages of the appliances were its cheapness, its portability and ease of manipulation. At least during the war it might help out in the absence of the Bárány apparatus.

DISCUSSION.

Dr. Loewy was of the opinion that with two such apparatuses and a switch they could be used as a malingering test, just as was done with a noise apparatus.

Paper: Dentigerous Cysts, With Report of Case; X-ray Plates.*

By Ira Frank, M. D.

DISCUSSION.

Dr. Joseph Beck regretted that Dr. Frank had not spoken more at length regarding treatment. The treatment was interesting because recurrences were frequent. Before he adopted his present method of treatment he had nothing but recurrences. Scraping the teeth out with a curette was followed by healing, only to have the growth return. A small vestige of the epithelium would remain in some part of the cyst and a recurrence would take place. The treatment which had given him absolutely perfect results was as follows: In opening the cyst the cavity was thoroughly scraped with a curette, the whole lateral and anterior wall was removed with forceps and then the burr was used to remove the surface of the bone so that there were no remains of the epithelium. Then the cavity was filled with a mixture of spermaceti, wax and iodin—33 per cent spermaceti, 66 per cent paraffin, and enough iodin—about one-half of 1 per cent—was the mixture which was used originally by Moroff in healing cavities of the leg. This was packed in tight in the cavity, and the mucous membrane of the lip side was wired to the surface above the teeth. The wax was allowed to remain in, and it could be seen to be extruded until the cavity was filled up. There had never been any recurrence since adopting that treatment. In the case of the boy he exhibited, he expected to adopt another method, which he thought would be even more satisfactory. This was based upon some experiments he was making with a mixture of ground up cartilage, wax and iodoform. The costal cartilage contained a substance which was eminently satisfactory for filling out a cavity—more so than the inert substances.

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*See page 991.

One of the most interesting cases he had had was that of a lady who had a dentigerous cyst which was drained and washed and recurred for two or three years. X-ray examination showed a tooth high up in the antrum. At the time of operation he had great difficulty in getting the tooth loose, as it was very firmly imbedded. He took a chisel and pried it out and the tooth disappeared; it could not be found anywhere. He thought it might have been swallowed, but what she complained of was that her nose had become blocked. The following day she could not breathe through the side of the nose which she was operated on. On the third day an examination was made, some adrenalin being introduced, and there was the tooth in the inferior meatus. The interesting part was that between the nose and the cyst was the antrum; he had opened the antrum, and while there was a little blood in the antrum the tooth was not there, but had gone through the antrum into the nose.

In another case there was a cyst of the inferior maxilla. The patient was a little girl, and there was a tumor on the side of the face which was an adenomatous affair. That cyst was treated in a similar manner, with perfect recovery. Usually there is great danger of weakening the jaw, as the cyst absorbed the bone, and spontaneous fractures had resulted as a consequence. As a rule, more teeth were distorted in a cyst which was high up, and the teeth were usually not teeth but remnants of teeth.

Dr. Otto Freer described a dental cyst which he removed in 1913. The patient was a young woman of eighteen. For a year a swelling had been growing under the right half of the upper lip and under the right half of the nose, uplifting it. Examination showed a cyst which uplifted the nasal floor in the region of the premaxillary bone and lay above the right incisor and cuspid teeth of the upper jaw. Pressing upon the cyst caused fluctuation in the mouth in the gingivolabial fold. The diagnosis was a dental cyst originating from the roots of the right upper first bicuspid tooth which had been devitalized and crowned. An incision along the right gingivolabial fold exposed the thin walled cyst, which was dissected out from this cut upward into the nasal cavity. The cyst was removed entirely, although very frail and adherent to its surroundings. Care was taken not to seize the cyst in the dissection but only its surroundings, and in this manner rupture was avoided. Inspection of the bed of the cyst showed that soft as the cyst was it had created absorption of the right premaxillary bone and of the incisor crest under the cartilaginous septum. This left the anterior inferior border of the cartilaginous septum without support or attachment, so that it sprang into the right external nostril and had to be removed by a resection to clear this naris. The result was perfect breathing and the disappearance of all deformity.

Dr. H. L. Pollock stated that all the teeth he had seen had no fangs but simply a crown of the tooth. In pictures they appeared as if imbedded in the orbit, but taken stereoscopically this was shown not to be the case. In one patient, a boy of eighteen, the third molar had not erupted, so it might be taken for granted that it was an interrupted molar on that side. The case was to be operated in the same way Dr. Beck had described, and he thought there would be no recurrence if the burr was used so as to be sure to remove all mucous membrane.

Dr. George Paull Marquis was very glad Dr Beck brought out the point about the burr. His experience had agreed entirely with that of Dr. Beck's as regarded recurrences and in the future he would certainly use this method. He thought the cases really were divided into two groups, from the anatomic situation: those which lie anterior to the first bicuspid and those which were posterior. Those anteriorly situated were the ones which caused almost no trouble; it was a very simple proposition to eradicate those cysts. Those which lie posteriorly were more closely in association with the antrum and would cause more trouble; it was in those cases where the antrum was most likely to be involved and one would have to open into it.

Dr. Joseph Beck said the advantage of the burr was that one could get down to the mucous membrane of the antrum without entering the antrum. He would consider it a failure in the technic if he got into the antrum. In the case in which he opened the antrum he did not pack with wax, but treated it as an open wound and subsequently used bismuth injections and got a complete recovery.

As to the congenital side of the question, he had X-ray plates of a father and daughter who had dentigerous cysts. He operated the father fourteen years before the daughter. In looking over the work of Killian, he spoke of the dentigerous cysts in the nasal cavity, and one of the earliest cases he reported was such a cyst which went into the nasal cavity and included it.

Replying to a question from Dr. Frank, Dr. Beck said that recurrences usually were noted two or three months after the closure.

Dr. Ira Frank, closing, said his patient had been free from recurrence for about a year. The cyst in his case was removed by making an incision on the buccal surface of the superior maxilla, similar to that employed in the radical antrum operation. The mass was exposed and incised, after removing the teeth, which were firmly imbedded, and the cyst was easily shelled out.

Paper: The Intranasal Operation for the Relief of Chronic Dacryocystitis.*

By Carl F. Bookwalter, M. D.

DISCUSSION.

Dr. Frank Brawley stated that he had done some of these operations, and the modification he had made was to use the burr instead of the chisel, which made a less cumbersome and more rapid operation, and it was equally effective in removing plenty of bone. He thought it was an operation which there was not much opportunity to do. He had found it somewhat difficult to persuade a private patient that it was the operation he should have done. He believed the use of the burr was an improvement.

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*See page 982.

Dr. Joseph C. Beck said he had done about fifteen of the operations by the various methods suggested, the Yankauer first. He had seen his operation and brought home his instruments and persuaded a friend to let him operate on a case he had been probing. In this method he pried off the inferior turbinate with the curved hooks. That case was a failure, as was every other case he had done. In adopting the upper avenue a very small wound through the slit was necessary, and they used the Hollow modification of the operation, trying to line the cavity so there would be no raw surfaces. It had been their experience that in using the sounds after the operation the parts had contracted down and there was the same trouble. He thought the principal point in Dr. Bookwalter's paper was the wide cut, and he felt that he had not paid enough attention to that. He would try this method in any future cases. It seemed rational to drop the bottom out of the sac, pack and let it drain through the nose.

Dr. Robert H. Good said the pain could be relieved entirely by putting cocain into the sac and then injecting novocain around the sac. He had exhibited a patient about six months ago in which he did the combined West and Yankauer operation. He chiseled away the bony wall of the duct and sac, and then slit up the nasal duct and sac all the way, and the result was perfect. He thought if the bony wall was taken away and a good opening made in the sac, some day the duct might open up by itself and functionate because of the bony wall having been removed. Yankauer's point of relieving the pressure on the lower end of the duct by removing the nasal side of the bony wall was a very important one and should not be lost sight of.

Dr. H. R. Boettcher stated that he and Dr. Woodruff had five cases in which they incised the outer wall and took a burr and burred through and then sewed it up again. The immediate results were fine, and two did fairly well, but the rest closed up again.

Dr. Bookwalter, closing, said he had not had much experience with the burr, but had used the chisel to remove the bone. He should like to try the burr, not because he could do any better work, but the sound of hammering was disagreeable to the patient.

As to anesthesia, he had used cocain and novocain, injecting novocain into and around the sac, and while he spoke of pain, the patients had never groaned or cried out with pain. They always had morphin beforehand.

He thought the opening into the nose must be made large; quite a little of the nasal process must be removed. There should be a window in the mucous membrane just about the same size as the permanent opening was intended to be. The only instrument he ever passed through the canaliculus was the smallest probe. It acted as a guide as it came into the nose.

He considered the after-treatment very important. The patient should be seen two or three times the first week, twice the next week, once the following week, and kept under observation for several weeks. He had cases that had been operated upon from one to three years ago who were perfectly well and where he could demonstrate the opening with edges that had remained exactly the same for at least a year. He considered these cured, but of course there might be recurrences even after that. He had not had any recurrence of suppuration, and it would be four years in January since the first operation was done. He had had some with tearing, where there had been a slitting of the canaliculus or previous injury to it, but if the canaliculus were uninjured there was no tearing after the operation. He had done several of the operations at the Eye and Ear Infirmary and the rest in clinical and private practice.

SOCIETY PROCEEDINGS.

NEW YORK OTOLOGICAL SOCIETY.

Meeting of January, 1917.

Acute Hemorrhagic Otitis Media.

DR. EAGLETON said he wished to report a case which he imagined was more common than they realized, and yet he had never seen such a case nor heard of it. It was a case of acute hemorrhagic otitis media, causing acute necrosis of the mastoid with metastatic thrombi in different parts of the brain and a general meningitis, followed by a general sepsis and death in four days. The reason he thought these cases were not described was because they might have been overlooked, and the case had been questionable as to whether it was in reality an ear case.

The patient was a man who looked to be about seventy years of age, but whose age was given as fifty-six when admitted to the City Hospital on December 21st.

In the history given by his wife, she stated that the patient was perfectly well, had never had an earache or discharging ear, until four days from admission, when he returned from work with a severe pain in the left ear. This continued to grow worse, and he became irrational during that night. The following day he was unconscious and had remained so since. Two days before admission to the hospital he had repeated chills.

When seen at the City Hospital the man was in deep coma. There was a hemiplegia of the left side, involving the arm, leg and face. Involuntary movements of the right side of the body, a strong bilateral deviation of the eyes to the right, a slight nystagmus; haziness of the right cornea from exposure had prevented an ophthalmoscopic examination of the fundus, but the left fundus showed a slight papilledema. The left drum membrane was not intensively red, but had lost its color, and the whole posterior quadrant was hanging down as in mastoid. A paracentesis was followed by a large quantity of bloody serum. Following the paracentesis Dr. Eagleton was of the opinion that the main trouble was the result of the general sepsis and not of the ear trouble.

Cerebrospinal fluid examination showed fluid under pressure, slight amount, cloudy and full of bacteria, almost like a culture tube. Dr. Harrison Martland said it was probably a pneumococci, although certain strands looked as if it might be a streptococci mucosus capsulatus, or a pneumococcic infection.

The man died on the night of the paracentesis, on the fourth day from the beginning of his illness. Examination of the head showed between the arachnoid and dura a large amount of pus, differing from the ordinary cases, in that the pus was not alone in the subarachnoid spaces, but was free in the subdural space. The pus was present in such quantities that one could wipe it away with a knife.

Scattered through the substance of the brain were many areas of encephalitis from the deposit of infective thrombi, these areas being surrounded by an area of edema. In the internal capsule of the right side there was a rather large area of encephalitis from an infective thrombus. The cerebellum showed numerous hemorrhages into its cortex, such as is seen in sepsis from a suppurative endocarditis.

The left mastoid was blackish from acute necrosis, with hemorrhagic infiltration of the substance of the bone and of the mucous membrane. The middle ear was full of clots, which may have been the result of the paracentesis that occurred only a few hours before. The walls of the mastoid were full of a gelatinous serum, as seen in very rapid sepsis.

A complete postmortem was made of all parts. There was a purulent bronchitis, and lungs giving no evidence of pneumonia. The heart was normal. The stomach had undergone antemortem digestion of the mucous membrane.

The interesting point of the case was that following the acute otitis media there resulted an acute necrosis of the region around the middle ear and mastoid, followed immediately by infective thrombi in the terminal vessels of the brain and a purulent meningitis. Then followed of course general sepsis The case was important because Dr. Eagleton thought that they were probably more common than had been realized. Dr.

Harrison Martland had stated that he was very positive that this was a case of primary infection of the ear of mucosus capsulatus.

DISCUSSION.

Dr. Phillips said, regarding Dr. Eagleton's patient, that he could not say he ever had a case with exactly the same history, but had seen cases of very rapidly developing meningitis, with fatal results in from two to five days. He thought it was a pretty rare condition.

Dr. Dench said that he had never seen, so far as he remembered, a case of streptococcus capsulatus infection complicating a hemorrhagic mastoiditis. He saw a number of cases of hemorrhagic mastoiditis—from three to four each year—but he did not remember a single case of this character in which the infecting organism was a streptococcus capsulatus. Cases of mild otitis media with fatal meningitis are not uncommon. He remembered a case of this kind occurring at St. Luke's Hospital several years ago, in which the middle ear symptoms were very slight, amounting to simple congestion along the periphery of the drum membrane and along the hand of the malleus. The patient had a high temperature on admission, and was operated on as an emergency case the next day. The patient died immediately after operation. This case was not a case of hemorrhagic mastoiditis, however. He had always considered that here the middle ear infection and the meningeal infection were concomitant and not interdependent. The speaker had seen a number of cases of streptococcus capsulatus meningitis, but these had always been insidious in their onset. He remembered one case of extensive mastoiditis, not of the hemorrhagic variety, with a sinus thrombosis necessitating removal of the internal jugular vein. This patient was about to leave the hospital at the end of three or four weeks after the operation, when, without warning, he suddenly developed a high temperature and became unconscious. Streptococcus capsulatus was found in the spinal fluid, and this patient died less than forty-eight hours after the onset of his meningitis symptoms. This was probably a case where the organism had become incapsulated and suddenly, as the result of some unknown cause, had been liberated into the cerebrospinal lymph space. Reasoning from analogy, one might think that infection of the meninges would occur more frequently with the hemorrhagic variety of mastoiditis than with any other variety, as a small tributary vessel might easily become occluded by a clot and this clot might extend backward toward the meninges and cause infection.

Dr. Haskin asked if there was a cephalitis in this case.

Dr. Eagleton said that in the postmortem examination one could see areas where infective thrombi had lodged in different parts of the brain in the internal capsule. There was a hemiplegia of the same side, and, of course, if the man had lived long enough, he would have had an abscess. The infective thrombi was the point of discussion—as to whether an infection could begin in the morning and that night a patient become irrational, followed the next day by coma. It was an interesting point—whether the vessel had been plugged by infective thrombi. If the patient had lived long enough there would have been multiple abscesses of the brain. This was practically in contradistinction to what Koerner had stated, that all these cases were by direct extension. The speaker was sure, from postmortem experience, that direct extension to the brain, in brain abscesses, was a very rare thing. An abscess generally results from a retrograde thrombosis. Of course, we have extradural, intradural and intracerebral abscesses. Both extradural and intradural abscesses were by direct extension, but a large proportion of intracerebral abscesses were from retrograde thrombosis, the result of the plugging of a vessel.

Dr. Berens asked if there had been anything about this man, in the postmortem examination, that could possibly have been thought mastoid and brain symptoms, which could have come from metastasis. Dr. Eagleton had spoken about the stomach being predigested. The speaker asked if there was any focus in the body one could have gotten metastasis from.

Dr. Eagleton said that Dr. Martland thought that predigestion of the stomach wall was not an uncommon thing.

Dr. Haskin asked what the condition of the pancreas had been. This condition, in diseases of the pancreas, was very apt to occur. The fluids of the pancreas, escaping into the peritoneal cavity, caused this necrosis.

Dr. Eagleton said the pancreas had been examined, but he had no notes of its condition.

Dr. Haskin said he could not see from the blood current how one got metastatic conditions in brain abscess from the lateral sinus, which goes out of the skull and not into the brain.

Dr. Eagleton said that at the tegmen tympani there were a number of small vessels, any one of which might become thrombosed. The peeled vessels of the cortex had free anastomosis one with another, so that if a retrograde thrombosis reached the cortex, it caused no nutritional disturbance; but when the vessels in the substance of the brain became thrombosed, they being terminal, from nutritional disturbance, necrosis resulted and an abscess formed. Of course, abscesses that were extradural were by direct extension, but in those inside the brain substance, as he had opened it, the brain looked perfectly normal.

Dr. Eagleton said why he happened to speak of this case was because of the rapid death, and it showed that if the man had lived long enough he would have had brain abscesses.

Dr. Haskin said he did not see how extension could come there through the blood vessels of the tympanum. This was distinctly separate from the brain, and the blood vessels went into the lateral sinus from the petrous sinus. The speaker asked how one was going to get direct extension through this. He said one might probably get an embolus there, but not a thrombosis.

Dr. Eagleton said they did not want to speak of embolus, but of vesicular occlusion, which is caused there from a thrombosis or embolus. This frequently happened in filled veins.

Closure of Old Mastoid Wound.

Dr. Phillips said this case was rather new to him, although he thought that the primary part of the work was common and had been performed by all men in this line of work. The patient, a man, had a very deep sulcus, following an acute mastoid, and instead of the granulations coming out toward the surface, the skin had crept inward. The man had a very deep depression, and a smaller, deeper hole in the antrum region, but no communication with the middle ear.

He came to Dr. Phillips to ascertain if he could get rid of this cavity in his mastoid process, and the speaker told him that it could be done. Over a year previously the mastoid operation had been performed in California, and the bismuth paste was used a few days after the operation and healing had taken place. The patient said there was a discharge for a long time, and he had seen particles of the paste coming out, and was quite positive a good deal had come away.

In attempting to get to the bottom of the old wound, and in order to elevate the skin, Dr. Phillips had unavoidably entered the mastoid antrum. He had a good opportunity to study an antrum following the use of the bismuth paste. As a matter of fact, there was no solid material found, and the cavity was filled with what appeared almost like a membranous material that had no particular significance. The wound being apparently sterile, he used the blood clot, allowing the wound to fill full of blood then stitched it up.

The subsequent history of the case was the interesting part When the patient came out of the ether he began to expectorate blood quite freely—not an enormous amount. This continuously for about four days. Knowing that this blood came from the antrum by way of the eustachian tube, Dr. Phillips had feared it would serve as a pathway of infection to the old wound. The drum had remained intact, but was dark colored for several days. The wound healed promptly, and there is now absolutely no depression behind the ear. Two months had elapsed since the operation.

DISCUSSION.

Dr. Harris said he saw a very remarkable X-ray picture, which Dr. Law had shown him. The man came into the clinic with a diagnosis of fracture of the skull with a swelling over the occiput. He was sent to the radiographer for a picture. Dr. Law said the man had told him that when he blew his nose the swelling at the back of the head bellied out. Dr. Law made him out as a candidate for a lunatic asylum, but when he saw the man's picture he thought the man was nearer the truth. There was a remarkable development of the frontal sinuses and ethmoidal cells. There was a completely pneumatic mastoid, posterior and anterior, directly back to the occiput. It was perfectly possible that when the man blew his nose he filled the eustachian tubes and antrum, and through these cells he was able to extend or distend the swelling.

Dr. Phillips said that for two or three days afterwards the drum looked a little bit congested, but it never broke down. The man now heard as well as he did before.

Dr. Haskin said he had seen the patient to which Dr. Harris referred, at the hospital. The swelling was as big as a fist, and it sometimes took forty-eigth hours for it to disappear. It ballooned out on blowing the nose hard, but when Dr. Law first asked him to see the patient, he thought it was an angioma. The patient had very thick dense hair, and the fracture of his skull had taken place in childhood, about seven or eight years previously. It was rather remarkable that he did not get an infection. It was interesting to know what would happen if the cells became involved.

Dr. Dench said that in regard to the plastic operation reported by Dr. Phillips, he had used this method on several occasions where it had been necessary to operate for an unhealed mastoid wound, and that he had obtained excellent results. He did not consider, however, that they were true blood clot cases. He believed that the good result was obtained by the obliteration of the operative cavity by approximation of the edges of the wound, rather than by filling the cavity with a blood clot and allowing this to organize.

Dr. Christian R. Holmes, in an article read before the International Otological Congress in Boston, had made a very valuable suggestion in regard to the treatment of all cases of mastoiditis, and that was, that the periosteum should be sutured separately in these cases. If the periosteum is first sutured and then the superficial wound closed, and pressure is made over the wound, the soft tissues sink into the cavity resulting from the operation and obliterate this. In this way all dead spaces are done away with, prompt healing takes place, and there is a minimum amount of deformity. This form of procedure is really a modification of the procedure suggested by Dr. George Leland, who advised that in the mastoid operation a flap of periosteum should be tucked down into the antrum, thus closing off this area. In none of these cases spoken of by Dr. Dench was the wound closed completely. A small drain was placed in the lower angle of the wound, and this was rapidly shortened at successive dressings. The method was really one of obliteration and not of filling it with a blood clot and allowing this to organize.

Mastoid With Acetonuria.

Dr. Phillips said he had a case under his care, in a child, that began with an attack of grip six weeks previously. The patient was taken ill with a cold, and very fortunately the physician attending her was a watchful man, who commenced to examine the urine. He found the child had an acetonuria, which he had great difficulty in getting under control. Both ears began to suppurate after two weeks. The doctor had succeeded only a few days previously in getting the urine in proper condition, and then only by using enormous doses of bicarbonate of soda in high enemas. Finally, on the Sunday previous, the mastoid had become so much involved that Dr. Phillips was obliged to operate. There was at that time a temperature of 103°, and the operation was followed by a reappearance of the acetonuria. The struggle again commenced, getting the child in proper condition. It was a staphylococcus aureus infection.

Bilateral Abducens Paralysis.

Dr. Dench said that he would like to ask how many of the gentlemen had seen cases of bilateral abducens paralysis. He had seen two such cases within the last two weeks. In one case a radical operation with a subtemporal decompression had been done at another hospital. When the patient was seen the entire radical cavity had become obliterated by the sinking of the intracranial structures into the middle ear. The operation was difficult on account of this sinking of the intracranial structures, but by careful dissection the overlying soft tissues were separated from the dura, and then, by means of a brain retractor, the dura and its contained structures were elevated and the radical cavity was cleaned. In the course of a few days the abducens paralysis upon each side was very much better. The patient had had considerable vertigo, but this disappeared rapidly after operation. The spinal Wassermann reaction in this case was negative. Two weeks later Dr. Dench saw the second case of double abducens paralysis, with facial paralysis upon one side. This patient had simply a mild acute otitis media on the side of the facial paralysis. In this case, also, the spinal Wassermann was negative. While the first case would be explained as a symptom of increased intracranial pressure, the second case did not allow of this explanation.

DISCUSSION.

DR. HARRIS said that Dr. Dench's case suggested one he had seen only a few days previously, which was equally puzzling, and that was one where the abducens paralysis was on the side of the opposite ear, and none on the affected ear side.

DR. HASKIN said they had a case at the Manhattan Eye, Ear and Throat Hospital with a double abducens paralysis. This man had always had trouble on one side. There was recently a diagnosis of some central lesion in his condition of bilateral paralysis. He had had paralysis on one side since childhood, but the other had come on within six months.

DR. DENCH said that his explanation was that in the first case there was probably a low grade of meningitis, and that the decompression operation relieved the pressure upon both sides. This would explain the improvement in his first case. A year ago he had reported a case before the American Otological Society, in which an abducens paralysis appeared suddenly following a mastoid operation with exposure of the dura. Here a larger exposure of dura, with elevation of the dura from the superior aspect of the petrous pyramid, caused a cessation of pain and a disappearance of the paralysis. These two cases were easily explainable, either as the result of a localized meningitis at the base of the skull, which accounted for the paralysis on one side, while increased cranial pressure might so crowd the cranial contents of the opposite side as to cause paralysis of the opposite abducens. The abducens nerve runs for a considerable distance in the skull quite unprotected, and for this reason is extremely liable to be interfered with, either from inflammatory exudate or from pressure due to increased intracranial pressure. Double cases could easily be explained by a broad neoplasm involving the nerves at the base of the skull, but there was no evidence of any neoplasm in either of his cases of double abducens paralysis. The second case of double abducens paralysis, complicated by a mild otitis media upon one side, admitted of no explanation as far as he knew.

Dr. Eagleton said that a double abducens paralysis was a very common thing in brain tumors. He said that an explanation of the double abductor paralysis in brain tumors is that from the internal hydrocephalus the sixth nerve is pressed against the internal carotid, and so paralyzed. The sixth nerve had quite a long course.

Dr. Haskin said that the sixth nerve would be in the cavernous sinus at this point.

Dr. Dench said that two or three years ago he had a case which showed that there was pressure upon the right vestibular nerve. At autopsy a cyst was found on the left side of the cerebellum which, by pressure, crowded the adjacent structures over to the right side, compressing the eighth nerve against the margin of the auditory foramen, thus accounting for the involvement of the right vestibular nerve.

Dr. Haskin said that the sixth nerve pierces the dura half an inch below the petrous ridge on the back of the sphenoid, enters the inferior petrosal sinus and passes through the dura into the cavernous sinus, where it lies on the outer side of the carotid artery. He said the third and fourth also lie on the outer side of the cavernous, but go over the top of the petrous ridge. The nerves lie on the outer side of the carotid artery, and Dr. Haskin did not see how pressure could be brought on the carotid at that point. It was more apt to be where it pierces the dura and enters the inferior petrosal sinus.

Dr. Dench said that abducens paralysis was very easily demonstrated, while the fourth nerve paralysis was not so easily demonstrated. Moreover, the sixth nerve runs for a considerable distance in the cranial cavity unprotected, while the course of the third and fourth nerves is comparatively short before entering the cavernous sinus. Owing to its unprotected position and its long course, the sixth nerve is particularly susceptible to pressure. Moreover, the blood supply of the sixth nerve is very limited, and an occlusion of its nutrient vessel might easily cause either impairment or complete abolition of its function. In the case already mentioned and reported by Dr. Dench at the last meeting of the American Otological Society, the paralysis seemed to be due to an inflammatory process spreading from the area of dura exposed at the time of the mastoid operation along the basal dura, thus involving the sixth nerve in the exudate or interfering with its blood supply. This inflammatory exudate also involved the dura covering the gasserian ganglion, and from this involvement the patient had excruciating pain over the distribution of the fifth nerve. As soon as these adhesions were broken up the sixth nerve paralysis disappeared, and the trigeminal neuralgia also disappeared. Not a drop of pus was evacuated during the operation, showing that the hypothesis regarding the involvement of these nerve structures in inflammatory exudate was correct.

Furunculosis With Capsulatus Infection.

Dr. Hopkins said that two or three weeks previously, at the Eye and Ear Infirmary, a man had a furunculosis which had been there two or three weeks at the time the case came under particular observation. He had a smear taken, which showed the streptococcus capsulatus. The speaker said this was an entirely unusual condition for furunculosis, and the diagnosis of mastoiditis, made by Dr. Perkins, was endorsed, although there was no mastoid tenderness and no other indication of mastoid condition. The case fell to Dr. Hopkins to operate, and he opened the mastoid, which he found absolutely free from inflammatory conditions, being normal throughout, until he came to the antrum, which, when opened, showed blood welling up from the antrum very rapidly, indicating that the middle ear was filled with granulation tissue. Although no pus was found, the smear taken showed a streptococcus capsulatus infection. The speaker thought it one of those cases which would suggest that some of the rapid cases of capsulatus infection of the brain might be due to such a condition, the bone itself not involved, but the capsulatus infection spreading through the middle ear into the brain tissue.

DISCUSSION.

Dr. Dench asked if a blood culture had been made in that case.

Dr. Haskin asked if a radiograph was taken of that case. He said he asked that question of Dr. Law, who had shown him a plate and asked him what he thought of it. It looked as if the whole mastoid on one side might be full of granulations and inflamed, although no area of pus could be seen. The case was one of very intense dermatitis with a great deal of edema and swelling over the back of the mastoid. Dr. Law and Dr. Rae had the case radiographed just to see what one would get in such an edematous condition. It was a picture with nearly every indication of mastoiditis, except that the middle ear was apparently normal. There had been no evidence of inflammatory condition in the ear. The thing all subsided under local applications. A radiograph taken later showed an absolutely clear mastoid. To all appearances the first would have made one think the whole area was involved.

Dr. Berens asked if the thickened condition on the outside was due to the furuncle in Dr. Haskin's case.

Dr. Haskin said there was in his case no furuncle of any sort.

Dr. Dench asked if in Dr. Haskin's case the outline of the mastoid cells came out clearly, and if the mastoid was opened.

Dr. Haskin said the mastoid cells were there and looked rather hazy, as though they were thickened. The mastoid was not opened. They took another plate, which showed absolutely as clear as the other side, after treatment.

Dr. Dench said that he had seen radiographs of a good many cases of severe acute otitis media. . In any severe acute otitis media the mastoid cells would appear cloudy. Subsequent plates would show a clearing of the acute inflammatory process and the septa of the mastoid cells would come out perfectly. Ordinarily he looked upon a radiograph as a very good measure for determining whether or not the mastoid cells were involved sufficiently to demand operation in cases of acute otitis media complicated by a furunculosis. A series of plates taken at intervals in such a case would show a gradual clearing up of the mastoid infiltration, in spite of the edema behind the ear due to the furunculosis. If the mastoid cells still remained cloudy, it was pretty certain that the operator had to deal with a mastoiditis as well as with a furunculosis.

Dr. Haskin said one would not expect a mastoiditis to clear up within four or five days, as in this X-ray plate.

Dr. Dench said one plate might have been good and the other a bad plate. One would have to have a tremendous amount of skin infiltration to hide the cells in the mastoid. He could not understand how it would occur in an adult type. They probably had a poor plate in the first case and a good one in the second.

NEW YORK OTOLOGICAL SOCIETY.

Meeting of March, 1917.

Presentation of Specimens.

Dr. John R. Page: The specimens shown were taken from a child two years old, which had been admitted to the hospital with a brief history. Two days before admission she was taken to the Presbyterian Hospital, with a high temperature, and was referred from that hospital to the Manhattan Eye, Ear and Throat Hospital, where a double myringotomy was done by the house staff. The child was admitted to the ward for examination and ran a temperature not higher than 100° or 101.5° for three or four days after admission, with quite a profuse discharge. On the fourth day, in the afternoon, the temperature went up to 102.8°.

Dr. Page said he was not at the hospital that afternoon, but saw the child the next day. He advised an operation, which could not be performed then, as the operating room was busy. He called up the next day to ask how the child was, and found that the temperature had come down and stayed down, not going above 101°. As there were a rather large number of operations to be done that day it was suggested that the case go over, and instead of being operated on the fourth day, there was no operation until the seventh day after admission, and probably the ninth day from the beginning of the trouble.

The temperature in the afternoon, just before operation, went to 104°. A double mastoid operation was performed and fairly well involved mastoids were found. A blood culture was taken during the operation. It was reported positive hemolytic streptococcus. The child's temperature then continued high, but after operation it varied between 101° and 103°, never above 103°, for five days. The wounds were very dirty and had a lot of green pus in them. There was exposure of the sinus on one side, which was very small.

Dr. Page thought the child was absorbing this pus through the sinus, and believed that by doing the dressings twice a day it might improve. When this was done the temperature subsided and stayed between 100° and 101°. On the twelfth day after operation the child had been sitting up in bed, with a temperature fairly low for six days, apparently doing well, and feeding itself, when it suddenly fell over. The patient went into coma that morning at eleven o'clock, with convulsions confined to the face, right arm and leg, spasmodic in type, lasting about two and one-half minutes, every ten minutes. The left pupil was dilated, the right smaller, but both were irregular. There was a double ankle clonus and Babinski, but no Kernig. The stomach and intestines were dilated. There were convulsive seizures three or four hours later, limited to the upper extremities. The child remained in coma, with a paralysis toward the end. There was a right hemiplegia, with occasional twitching of the arms and face. Death occurred on March 20th, three days after the sudden seizure.

The autopsy report by Dr. Dwyer showed a large tortuous vein through the dura, and large subdural hemorrhage on the upper left vertex, involving motor centers, a large extensive superficial necrosis and extensive edema of the right front part of the brain, invading motor centers. Markedly free from any thickenings of sinuses, either ethmoids, frontals or sphenoid. There was no clot or any signs of macroscopic infiltration. The inner wall of the left sinus was the same as the right; it was somewhat thicker than normal, but showed no clot. There was a small area of bronchial pneumonia of the right lower lobe. The brain areas were quite necrotic.

Dr. Dwyer said the condition was purely hematogenous He could trace no extension from the mastoid.

DISCUSSION.

Dr. Duel said he thought this a very important case, illustrating a fact which they all probably knew very well, but which was being more and more impressed upon them, that it was perfectly possible to get a streptococcus infection in the blood stream through an area in the sinus, without the formation of a clot and subsequent breaking down of the clot. Dr. Duel said perhaps some of the members would recall that six or seven years ago he had presented to the society a series of charts from the Babies' Hospital, pointing very pertinently to the fact that a widely vacillating temperature in the presence of a mastoiditis or middle ear suppuration, was not always associated with a sinus thrombosis

Since that time he had had a great many puzzling cases in which there had been great doubt about the question of operation. Many of them had recovered without operation. He thought that there was often a grave question as to whether these cases were actually showing an infection which might or might not be due to a sinus thrombosis. In the absence of a demonstrable bacteremia it was a question as to whether it was not more advisable to isolate the infected area from the general circulation, even though a thrombosis was not evident.

Dr. Dench said, in speaking on Dr. Duel's remarks, that he had in one case been inclined to take the view advanced by Dr. Holt. In this case Dr. Holt had been wrong, as the temperature was due to a mastoiditis complicated by a sinus thrombosis. Despite adverse opinion he had operated on this case twelve hours after he saw the patient, and found the mastoid full of pus. The temperature fell immediately after opening the mastoid, but later rose again, and the sinus was exposed. The sinus contained a clot. The temperature again fell after the removal of the clot, but inside of twelve hours rose again, and the internal jugular vein was excised. The vein contained a clot extending throughout its entire course. The child subsequently died of gangrene.

Speaking of other cases in which the sinus was involved, Dr. Dench pointed out that a parietal clot was the most dangerous condition to be found in the sinus. In one case showing signs of sinus thrombosis he had operated, and the sinus bled very profusely. Notwithstanding this the internal jugular was excised, the temperature immediately fell, and the boy made a complete recovery. A clot in the lateral sinus is an effort on the part of nature to shut off the focus of infection from the general circulation. When a clot breaks down we have symptoms of pyemia with localized foci of infection in the various viscera—that is, in the lungs, liver, spleen, heart, brain or intestines. The only chance of saving many of these cases is to cut off the primary focus of suppuration from the general circulation by prompt excision of the internal jugular vein. In many of the cases thus treated no demonstrable clot will be found, either in the vein or sinus. The wall of the vein and sinus, however, will be found to be infiltrated with pathogenic organisms. The infective process, in other words, extends from the local focus of suppuration in the bone to the wall either of the sinus or the jugular bulb, and then infection travels through the wall of the sinus until it affects the inner wall, with the formation of a parietal clot. In all cases the infiltration of the vessel wall extends beyond the limit of any demonstrable clot. Of course, the excision of both jugulars, when indicated, is naturally a very grave condition, as the cerebral circulation is apt to be greatly disturbed.

Operators should always remember that when a small area of sinus is exposed, this area should be intentionally increased until at least three-fourths of an inch of the vessel wall is exposed. With a small area of exposure the outside wall of the sinus would become infected through the infected field, no matter how carefully instrumentation is carried out. As a result of this local infection the sinus wall swells, and the circulation in the wall is interfered with by this swelling. The infection then travels to the internal wall of the vessel, giving rise to general systemic infection. If, on the other hand a large area of sinus wall is exposed, the circulation in the vessel wall is not interfered with by swelling of the wall of the vessel. The vessels of the sinus are able to take care of this local infection, and no general infection results.

Dr. Eagleton said the case he had presented at the last meeting was very similar to Dr. Page's case. The whole thing went through in four days, practically the same as this case. They were realizing that processes could be as rapid as that, and in forty-eight hours his patient, a man, was in complete coma.

Dr. Page said, in regard to exposure of the sinus, that exposure of the sinus had been made simply because the inner plate was cracked in one place and he feared something would get under the crack. It was widened so that there would be no retention.

Presentation of X-Ray Plates.

Presented by Dr. Haskin.

Dr. Haskin said he would like to pass around two or three plates. At the last meeting they had spoken of a case of pneumatic condition of the mastoid, and he thought it might be interesting to look at its development. The man had had a fracture of the skull when he was a child, having fallen on his head. He was now about eighteen years old and had a tumor at the back of the head, which could be felt on palpation, and when he blew his nose hard it would be enlarged. After this it took about forty-eight hours to go down again. They were not sure whether it was angioma, and inserted a large needle, getting an expression of air through the tube.

The second plate showed a peculiar form of microtia in a child ten years of age. The position of the lobe of the ear was down in the middle of his cheek. There was no development of the upper ramus and the condyle of the jaw on that side. There was a tremendous glenoid fossa. The teeth were in the process of the bone, but had never erupted. The X-ray picture showed a petrous bone, but no formation of a mastoid, and what was taken to be the superior semicircular canal could be seen. In the question of hearing, with the noise apparatus in the sound ear the patient undoubtedly could distinguish the difference between a loud ringing fork and whistle or voice, but that was all. There was absolutely no functionating static labyrinth, and it was merely of interest from the standpoint of atresia. Another was in a girl twenty-two years of age. There appeared to be no mastoid process back of the cells, but the canal seemed to be normal, except that at about one-half of its depth it was blocked off. There was no membrane in it at all, and no hearing whatever in that ear. She had measles when six years old and had never had any hearing. There was no development of the mastoid, and this was somewhat similar to the microtic case. This woman had a perfectly normal auricle but no ear. The third case was a man, twenty-eight years of age, with microtia.

Case With High Temperature.

Dr. Haskin showed a plate, which he said was interesting, because in the case Dr. Law had said the left mastoid was clear. The right mastoid showed involvement and sclerosis of the superior walls. The woman had an operation on the left mastoid seven years previously. Dr. Law was quite astonished and said it could not have been so. The woman had some deep cells away down between the canal wall and the sinus, and some over the antrum and knee of the sinus. The sinus stood out wonderfully clear. Dr. Law did not think there was any diseased condition in the mastoid, but on operation a very decided involvement was found. He thought it was nothing but a normal mastoid, but it had been very extensively excavated seven years previously. The subsequent history, Dr. Haskin said, was the most remarkable of any case he had had to deal with for a great number of years. The patient from earliest girlhood had been going to hospitals. She had been operated on for appendicitis, for tonsils and adenoids, a submucous operation, and then another submucous operation, which was unsuccessful. Within two hours after the last operation she complained of intense pain. The house surgeon attempted to do a paracentesis four times. She was then transferred to Dr. Haskin by Dr. Harris. The speaker looked at her drum membrane, which was red. He incised the drum membrane, but found no pus at the time. The intense pain kept on for three or four days after the last paracentesis was done. Finally he operated on the mastoid and found it extensively involved. After elevating the skin and soft tissues, an area of black granulation tissue was exposed, extending down into the antrum. The dura was slightly exposed over the tegmen of the antrum. He cleaned out all the walls from the knee of the antrum and took out the tip, which was full of new bone, which had formed from the previous operation. Before, the surface had simply been taken off without the deep tip cells. None of the cells which Dr. Law spoke of were involved. They curetted the region between the sinus and canal wall and finally uncovered three or four perfectly large sized cells.

The patient was put back to bed with no sutures, and they expected the pain to be relieved, but it was not at all. She could not be touched anywhere over the head without pain. Dr. Haskin presented her temperature chart to the society, taken during the time she remained in the hospital, and a report of what was done for her. She had a negative Wassermann reaction. Both cerebral, spinal and blood cultures were negative. The complement fixation test for tuberculosis, negative. Four blood cultures had been taken which were all typhoid and paratyphoid were all negative, likewise the reflexes and eye grounds. The abdominal reflexes were slightly increased.

Dr. Haskin said they had watched her very anxiously, and finally the family were so anxious to have something done that they took her away. Meanwhile the wound was healing up very nicely. She went away from the hospital on February 25th and came back on March 6th with severe pain. The interne who examined her said the physical examination was negative. They thought of meningitis after the high temperature. After she had been in the hospital from the sixth to the eighth, the temperature began to go up suddenly. It went up to 104°, 105°, 106°, 107° and 107-2/5° and then dropped back to 106°, when she left the hospital. There was absolutely nothing found to account for it. Dr. Rae and Dr. Friesner went over her and tried to find out what it was.

They did not do a lumbar puncture until the day she left the hospital finally. Ten cubic centimeters of perfectly sterile fluid were withdrawn and a normal saline reintroduced. With this high temperature the pulse rate was only 72 and 68, and then up to 78. This was a very low pulse rate, the speaker said, for that temperature. Five days after the operation on the mastoid she developed a partial facial paralysis. She could close here eyes, but there was a partial paralysis. The most interesting thing was that they took the girl away with a temperature of 106°. She was taken to the Neurological Institute, and Dr. Haskin presented the temperature chart taken during her stay there. In the Neurological Institute the temperature was normal and had been normal ever since admittance.

DISCUSSION.

Dr. Dench said that in a case of neurasthenia the eighth nerve might be involved, giving rise to severe pain, and in neurasthenic cases high temperatures, not explained by any known cause, were not uncommon. He remembered one case occurring in St. Luke's Hospital in 1885, where a patient would occasionally shoot a temperature of 106° and 107° for no assignable cause. The patient made a perfect recovery, and in this case no aural lesion was present. The diagnosis in the case of a neurasthenic could only be made by elimination. Practically no dependence could be placed upon the symptoms complained of by the patient. If any focal suppurative lesion were present, naturally the surgeon was greatly concerned. In the absence, however, of any positive results on cytologic examination, the mere presence of temperature alone in a neurasthenic should not be sufficient cause for operation. With normal blood and normal spinal fluid in such a case, the temperature should not cause undue alarm.

DR. HASKIN said he had brought the case up because he thought Dr. Rae would be present, who wanted to report the same kind of a case. When they did the mastoid the temperature began to run down. The lumbar puncture showed a sterile fluid. They had to give her one-half grain of morphin every three or four hours. In his case they did a decompression and also later explored the brain, and found nothing. The patient died, and they found a localized basal meningitis.

Another such case, Dr. Haskin said, which Dr. Dench might recall, had been presented by Dr. Packard before the Eastern Section of the Triological Society, entitled "An Error in Diagnosis." The patient was watched and everything had been done. They did a mastoid, a decompression, explored the brain, and nothing relieved the pain. There was a sterile cerebral spinal fluid, and the patient died. Miliary tubercular meningitis was found at the base of the brain only. In this case they had been absolutely puzzled and could find nothing at all except pain and temperature. Dr. Rae's case was exactly the same, and when he was asked whether the case presented should be operated upon, he said to leave it alone, as his case had happened two weeks previously. His patient had died with an entirely localized basal meningitis, and yet the cerebral spinal fluid was absolutely negative, both with chemical and bacteriologic tests.

Personally Dr. Haskin felt that his patient, the girl, had a localized basal meningitis, and it would be interesting to know the outcome.

He said his patient did not sleep, and Dr. Elsberg said they could only keep her quiet by giving her morphin. They gave her one-half grain of morphin at six o'clock, and another at four in the morning. She yelled so that everybody was disturbed, and yet during the day she was fairly comfortable. She had pain in the day but did not yell.

DR. DENCH mentioned in this connection a case seen in consultation, in which a very good radical operation had been performed. The patient complained of uncontrollable headaches. The Wassermann blood and spinal reactions were negative. The question came up whether a decompression operation would be of benefit. Dr. Dench did not advise the decompression, but said that he would perform the operation if no condition could be found which would account for the pain. The patient was sent to the family physician, who knew the family history. He administered antiluetic medication. The patient made a complete recovery, the pain disappearing absolutely. The family physician knew in this case there was a positive luetic history in the family.

DR. HASKIN said no lumbar puncture was done until one day before she left the hospital, but after the lumbar puncture she still suffered intense pain. The temperature dropped down to 102°, but when she left the hospital it shot up to 106°. Dr. Haskin said he did not think it at all impossible that the lumbar puncture was a source of relief and that the patient got sufficient relief after drawing off that amount of spinal fluid, which lightened up whatever was irritating the heat center, and in this way might have relieved the temperature.

Bilateral Abducens Paralysis.

DR. PERKINS said that at the last meeting of the society the subject of bilateral abducens paralysis from brain tumors arose, and Dr. Eagleton had given Cushing's explanation, as due to distention from accumulation of cerebrospinal fluid in the descending horn of the lateral ventricles, pressing upon the nerve as it passed between the cavernous sinus wall and the carotid artery. Dr. Haskin could not understand how this could take place without the third nerve being involved at the same time. Dr. Perkins said he had made several dissections on cadaver, and after doing them it came out perfectly plain. The internal carotid artery got into the cavernous sinus at the foramen lacerum medium, turned anteriorly and then made another turn upward, at the same time approaching the medium line. The sixth nerve is in immediate relation with it and the wall externally, so that pressure here would bring the horizontal part of the artery and external wall of the cavernous sinus still nearer together, thus resulting in interference with the function of the nerve—that is, abducens paralysis. The third nerve enters the cavernous sinus from above and does not come into such intimate relation with the artery, lying, as it does, in a much wider part of the sinus.

DISCUSSION.

Dr. Haskin said that the sixth nerve went below, into the inferior petrosal sinus, and then passed up. The third nerve passed over the ridge of the superior petrosal and lies in the cavernous sinus. The sixth passed underneath the ridge of the petrous, whereas the third goes over the superior border of the petrous. He said he still did not see how pressure on the carotid artery would cause the paralysis, and thought it was due to pressure before entering the sinus.

Dr. Perkins said that Dr. Haskin had claimed that the third nerve must necessarily be involved, and that the explanation given accounted for the escape of the third nerve. Dr. Perkins said the carotid artery could not be called a soft substance, compared with the nerve. It is a well known fact that slight swelling of adjacent tissues producing very little pressure on the sixth nerve causes abducens paralysis. The pressure exercised by an artery may be very great in its effects—for instance, the absorption of bone from aneurism. The vulnerability of nerves from pressure of arteries is strikingly shown in some cases of paralysis of the recurrent laryngeal.

Dr. Haskin said he also had made some sections which showed the third, fourth, fifth and sixth nerves where they pierce the dura and their course in the sinus itself.

ABSTRACT OF THE SCIENTIFIC PROCEEDINGS OF THE THIRTY-NINTH ANNUAL CONGRESS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

HELD AT ATLANTIC CITY, NEW JERSEY, MAY 28, 29, 30, 1917.
EMIL MAYER, M. D., ABSTRACT EDITOR,
NEW YORK.

The president, Joseph L. Goodale, after expressing his appreciation of the honor conferred on him, said:

This year we meet at a time when the thoughts of all are centered upon our entrance into the great war. Aside from the fact that lesions of the upper air passages are especially frequent in the present war, both from the character of the missiles and from the use of asphyxiating gases, observers have noted a great increase in inflammations of the nose and throat spread by contagion among the numerous masses of men in modern trenches.

The contributory causes are the prolonged stay in the trenches, the dampness and want of ventilation in the dugouts, together with the defective light, in which the virulence of microorganisms is increased, or at least the bactericidal influence of sunlight and fresh air is absent. These conditions appear to favor not only the spread of acute catarrhal infections, but also of cerebrospinal meningitis. The importance of stamping out foci of this latter disease has suggested the desirability of examining recruits with reference to the possibility of their being carriers through lodgment of the meningococcus on the mucous membranes of their nasopharynx. Disinfection of the mucous membranes of carriers has been successfully accomplished by placing the individual in a chamber containing a certain percentage of chloramine vapor for a few minutes. The procedure is said to cause but slight irritation to the mucous membranes, but seems to have almost a selective action on the microorganisms in qeustion.

Some sections of the line appear to be especially unhygienic, and numerous cases occur of infection which in ordinary life are rare, such as perichondritis of the larynx and primary laryngeal tuberculosis. Latent systemic disease is prone to be lighted up—such as tuberculosis and syphilis. Bleeding from the nose, trachea and bronchi, at times of an alarming extent, often occur. In albuminuria, fatigue and cold are apt to bring on glomerulonephritis, a not infrequent symptom of which is edema of the glottis. The unfavorable influences are increased by the difficulty of changing clothing, transportation in open vehicles, and by rapid changes of temperature.

Direct laryngoscopy has proved its value in locating traumatic lesions.

Secondly, may we not then profit by an examination of the writings of those who are now endeavoring to indicate some of the errors of judgment and conduct in matters of education and research?

In illustration of past deficiencies in this respect may be cited the fact that for many months the English authorities, while rigorously excluding from Germany such articles as saltpeter and cotton, nevertheless were permitting the exportation of lard from this country in large quantities, in ignorance that it is the source of glycerin, and consequently a prime requisite in the manufacture of explosives.

We must, therefore, recognize that as nations court destruction for want of science, so our profession in America runs the risk of being outstripped by that of other countries unless it rests upon an equally secure foundation of scientific training with all that this implies.

Another matter which I wish to present for your consideration is the slackening up of scientific work in allied countries in the early part of the war.

We should differentiate between those educators and investigators, on the one hand, whose position permits an advantageous deflection of their activities into channels capable of rendering a direct national service, and on the other those individuals who, by reason of their age, lack of physical qualifications, or from the nature of their work, may regretfully feel that they must stand aside without a definite service to offer.

Such men are capable of making a very real contribution to the nation. It is for them to uphold the best traditions of their past, to maintain without faltering their customary work of teaching and investigating, and to present to their comrades whose good fortune it may have been to participate more directly in military service, on the return of peace, a definite record of having kept undimmed each in his own chosen field the light of education and of research.

Further Observations Upon the Use of Radium in Diseases of the Upper Air Passages.

By D. Bryson Delavan, M. D.,
New York.

From a report by Dr. Ewing and Dr. Janeway of the Memorial Hospital in New York City, an institution devoted to the study of cancer, the writer presents a summary of one hundred and eighty-four cases of cancer of the upper air passages out of a total of four hundred and twenty-two cases.

The successful cases given in the report of cancer of the mucous membranes are illustrative of the best service that radium in the light of our present knowledge can perform. In the majority of them the lesion was small and probably operable, but for some reason operation was deemed inadvisable or was refused. While all show complete clinical retrogression, this—from the point of view of those who only consider as cured those patients who are well after three to five years—may not be an absolute cure. The proof must be left to time.

In advanced cancer, any temporary benefit from radium is usually overshadowed by the later progressive extension of the disease. No patient of this class should be given treatment which will be followed by more than transitory discomfort.

To summarize a few after-points made in the report which seem to carry special weight:

Kowledge of the use of radium is making steady progress.

The work of but one year in a single institution shows a marked advance.

As with surgery as applied to carcinoma, so with radium, for the best results are gained by the prompt treatment of early cases.

The question at once arises as to how long it will be before the use of radium will be proved worthy to supplant surgery.

DISCUSSION.

Dr. Charles W. Richardson, Washington, D. C.: I have had very unfortunate results after the use of radium in five cases which I recall to mind—four of cancer of the tonsil, and one of cancer of the cheek. They have all died. The cancer of the cheek was discovered very early.

Now, these and the other two cases, which were later, all died in the usual course of time.

There is, however, one thing to be said in favor of radium in the treatment of these cases, and that is the wonderful relief it gives of all of the disagreeable symptoms. As they went along their course these cases did not look like extensive malignant disease of the tonsils, as we usually see them. There was very little odor and the lives of the patients were fairly comfortable and free from pain. One died from hemorrhage, and the other three from toxemia, and this toxemia came on one week and the next week they passed away. It was all very quick, and they were apparently in good health except for the local condition. Until the time of toxemia they were interesting in that way, that they were all very much more comfortable, but they all four were fatal, and fatal in about the time they would have been without any treatment at all.

Dr. Robert Clyde Lynch, New Orleans: I have four cases in my experience now, one of which I believe is definitely well. It is a case that has apparently been cured—at least the patient has had no recurrence for eighteen months. He received no benefit at all from the action of the radium until the radium was put in the laryngeal cavity through a tracheotomy tube. Seventy-five milligrams of radium and screened over with a milligram of pure gum rubber was left in situ for eight hours, tied into the larynx by means of a catheter, and put in through a tracheotomy tube passed through the mouth, the radium slung between two strings, the lower string tied to the tracheotomy tube and the upper string to the teeth. In that way the radium was brought into actual contact with the diseased tissue. This was one of the cases that had a recurrence after dissection by suspension, and that recurrence has entirely subsided and the voice returned, and he is apparently well—that is, he has been well for eighteen months at least.

In another case operated upon with a third recurrence it was thought to be perfectly inoperable, and the application of radium to this area caused perfect and complete subsidence of every evidence of the recurrence. It is now six months since this application, and apparently there is not the slightest indication of any recurrence of this metastasis to the second side. In this instance again we were able to supply the radium actually to the site involved, and I wonder whether inability to secure any results in the laryngeal areas is not due to the fact that the cartilage must act as an interceptor to some of the rays, or influence the action of the radium within the laryngeal box.

Dr. E. Fletcher Ingals, Chicago: A patient came to me from a distant state with a growth in the pharynx running perpendicularly. It was about one and a half centimeters in its various diameters and about three or four centimeters long; that is, it extended from the upper pharynx down to the arytenoid cartilages. I could not tell how much farther. The patient came with a history of not having been able to swallow for some time, and was much emaciated and in an extreme condition. The effect of radium in this case was something marvelous; the growth simply melted away. At the end of about eight days the tumor had all disappeared, but there was a large ulcerated surface that represented the base of the growth; and the most remarkable thing was that the ulcerated surface completely healed so that within a short time one could not even see the scar. When this growth had disappeared I found that the patient still could not swallow, and then I was able to determine that she had cancer of the esophagus lower down. I passed a catheter down to the obstructed point, measuring the distance, and then placed his radium in the catheter and measuring the distance passed it down into the stricture. The result of the first application was to open up the stricture very decidedly, and a later application opened it up so that the patient could swallow very well. However, in one of the earlier applications the throat was burned so that it had the appearance as though it had been burned with boiling water or a hot iron. It became very sore and there was considerable sloughing. The patient had a great deal of discomfort for ten or twelve days; however, the burn finally healed.

Dr. J. Payson Clark, Boston: I want to add my testimony to what Dr. Richardson said in regard to the value of radium treatment in relieving the symptoms, especially the symptoms of pain. I was very much impressed with the relief which was obtained in swallowing by patients who had before suffered a great deal.

Dr. Lewis A. Coffin, New York City: I have had a little experience in the last year with radium. I had become thoroughly discouraged in its use and the effects from it. However, we are glad to get anything that adds a ray of hope to forlorn cases.

In one of my cases all the symptoms were very much relieved. The woman could not swallow at all when she came to me, and shortly afterward she was eating fairly and greatly relieved. But the glands softened up somewhat, and we thought it well to open those glands and let out the broken down tissue and put the radium in those glands and left it there all night. The woman, however, began to lose ground and she soon died.

Another patient had a growth in the epiglottis, and a pathologic examination showed it to be malignant. In that particular case it was just like adding manure to soil: the growth was very much stimulated and it grew more rapidly than if we had not used radium.

Dr. Burt R. Shurly, Detroit: I am enthusiastic enough in the use of radium to feel that it is our duty to at least use this method of treatment in all cases of malignancy where we so thoroughly understand that surgical methods are not sufficient to accomplish results.

I operated on one case which involved the soft palate, the upper jaw and the antrum from time to time—three operations in all—and after each operation there has been a very slow recurrence. During the past three years we have used radium at intervals of from three to six months, and the radium has been passed up into the antrum after the antrum operation and held there by the use of soft pliable copper wire which will bend in any direction.

Another case that I have had which has now been going on for about three years. By a series of applications of radium his case has undoubtedly been prolonged and been benefited, and what looked to be a very speedy fatal issue has turned out to be a slow process that has now continued for three years.

I do believe that radium has a very decided field of usefulness, but there is a great deal in the scientific application of it, and in the dosage and method by which it is applied, and I feel that it is a field within itself, and we should have an expert who will help us in that branch of the work and upon whom we can call at any time.

DR. EMIL MAYER, New York City: I would like to have the reader of the paper in closing give us some statement as to what class of cases have done best. It is very possible that we may innocently do some harm to the patient in our attempts at making a diagnosis. Ought we not, therefore, learn of the experience of those who have followed these cases and found that perhaps it may be that those cases have done best where the diagnosis has been made clinically, and no previous attempt made to remove a part of the growth for microscopic examination to prove the diagnosis? Is it not possible that that stimulates the recurrence of the disease? If we can have that made clear, it does seem that we may be able here to formulate some positive distinct rule as to the method we may adopt with safety.

DR. HARMON SMITH, New York City: I believe the good results that are obtained from the application of radium lies in the fact that practically all tumor growths vary in their virulency, and that those cases which are benefited by radium are less virulent than those cases which go on in their natural process of malignancy to a fatal termination.

I am associated with the same institution as Dr. Delavan, and I have sent a number of inoperable cases there—patients upon whom I had previously done a tracheotomy. This is an institution where every facility is available for properly applying radium in advanced cases, and each case went progressively on to death. In addition to these cases, I have sent two cases of retrolaryngeal fibroma, and one went on to death. In the other case there was such burning and excoriation of the mouth that we had to feed the man by rectum for a week, and instead of retarding the growth, it became accelerated and involved the cheek. This was an angiofibroma.

Dr. Norval H. Pierce, Chicago: Based upon his experience the speaker believes that while a growth may show the characteristics of carcinoma under the microscope, it may vary greatly in malignancy.

At the present time there must be an enormous number of these cases, and I cannot see that we have arrived at any particularly well defined opinion regarding the beneficial effects of radium, even at the present time. I can only state that my experience with radium has been in a broad sense disappointing.

Within the past two years I have had three cases of carcinoma of the superior maxilla involving the antrum, and without exception they have gone from bad to worse, although every means of applying radium has been followed, even to perforating the antral wall and putting a capsule of radium immediately into the growth. True, the pain has been diminished and the odor has decreased, but the disease has swept on as though nothing had been done.

I believe that if we depend upon radium for a cure in the early stages of laryngeal carcinoma, we will have about as much effect beneficially or otherwise, in the large majority of cases, as we would have if we depended upon faith. We only waste time.

I have had a case recently in which we split the larynx and applied the radium without cutting away the growth, a case of leucoplakia laryngis, where I had observed the patient for four years. This leucoplakia was situated on both vocal cords. The leucoplakia remained unchanged for four years. Suddenly from one of these placques on the right vocal cord, the posterior third, a swelling began—a fusiform swelling covered by mucous membrane—and a well marked cauliflower-like carcinoma developed. The larynx became very much inflamed, and the last I heard of him was that there was a probable recurrence of the growth.

The only possible chance for cases of carcinoma of the larynx is thorough removal by external means at a very early date, no matter how early. Whether we apply radium or not is immaterial; otherwise in extensive carcinoma the only means of safety is a thorough operation, and by that I mean laryngectomy.

Dr. James E. Logan, Kansas City: I believe if the suspension method is used and a local application of the galvanic cautery is made directly to the growth, comprehending or taking in all of the growth so far as is feasible, that we will have accomplished practically all that we could accomplish, if not more, than in an application of radium.

If we are to judge from the literature pertaining to the subject, there appears to be very little if anything in the use of radium for such cases.

Dr. Joseph Beck, Chicago: It is about fourteen years since I started the employment of what I thought was radium in diseases of the upper respiratory tract, and only about five or six years ago did I find out (and then reported with negative results) that practically no radium was contained in the capsule that I was employing. So far as I am able to learn from the literature on the subject, too little radium was employed, and then when an amount, say one hundred and fifty to two hundred milligrams, was left in situ for a period, it burned the structures beyond repair, so that the remedy was worse than the disease. My results are absolutely negative as to cures of deep seated growths; I will not even say of far advanced cases, but of those which I would ordinarily class as operable. When radium was employed in such cases the ultimate result was either laryngectomy in time to save the patient, or a fatality.

If Dr. Mayer would have us treat cases without touching the growth, it does not seem to me it would add anything. We will not know what we are treating. This does not appeal to me. If we do not have microscopic examinations of the growths we are treating, it seems to me the subject would not advance very far.

Dr. D. Bryson Delavan, New York City (closing the discussion): The work which is being done at the Memorial Hospital in New York is very far in advance of anything that I know of that has been done anywhere else, and for that reason I have taken the liberty of presenting this résumé of that work.

In this report I mentioned, the hospital gives you the results of its work along this line. It is the best they have been able to do in three years' time with a very considerable number of cases. When more years have passed they will have thousands of cases, but their attitude in treating these patients is purely experimental—time will give more definite results, or at least more conclusive results, whether better or worse.

Of all the cases treated, there are a total of twenty-two which are believed to have fairly retrogressed. That means twelve per cent of the one hundred and eighty-four cases. There are also seventy-nine cases which have improved, which is a total of about forty-three per cent. There are also seventy-nine cases unimproved. This makes a total of fifty-five per cent supposed to be improved, as against forty-three per cent unimproved. In cancer of the esophagus, stomach, etc., the statistics are just about the same.

The method must be adapted to the case, and the ingenuity of the one managing the case must play an important part in the selection of the means of application.

The device of Dr. Robert Abbe is simple, ingenious, and very effective. It is intended for the application of radium to the more remote recesses of the body, and is especially useful in the larynx and pharynx. It can also be introduced in any part of the upper nasal cavity, which is extremely difficult to reach by any other method.

There is another thing which has come to be used in the treatment with radium, and that is an obturator like a plate for artificial teeth, extending backward to the site of the growth and furnished with grooves in which the radium can be placed to the location desired.

As to the hopelessness of cancer of the throat—it is pretty hopeless. The cases all die. We do not get the true statistics of surgery of the larynx. You cannot get them—no one will give them to you; but if you should get them, you would have a pretty ghastly record. Perhaps it might, therefore, be just as well to exercise a little patience toward radium until we find out what it can do, and look forward with a ray of hope meanwhile to cheer the men who are working very hard to perfect it.

An Endolaryngeal Removal of an Unusually Large Lymphoangioma From the Larynx, With Complete Recovery of the Patient.

By Charles W. Richardson, M. D.,

Washington, D. C.

As is well known, this type of growth is very unusual and rare in the larynx, lymph angioma being rarer than the typical angioma. This growth presented a gross appearance, of a large bluish, mottled mass in the supracordal portion of the larynx, having its attachment on the right side from the false cord, the aryepiglottidean extending over to and covering the false cord on the opposite side. This was removed by endolaryngeal method, the writer having first tested the fact that it was not a hemorrhagic growth. On removal by puncture, it had a soft pultaceous feel and exuded a syrupy bloody fluid. It was five centimeters in length, three and a half centimeters in breadth, and two and one-half centimeters in depth. The pathologic investigation demonstrated it to be a true lymph angioma.

The paper deals with the method of operating in these cases, and also calls attention to the fact that this is the forty-second case of angioma, and its modifications, which have been found in the larynx.

DISCUSSION.

Dr. J. M. Ingersoll, Cleveland: I had the privilege of seeing this case before it was operated upon, and was very much interested in the report.

I think that it took considerable courage for Dr. Richardson to determine whether or not the growth was an angioma by incising it. I discussed the case with him then, and we both wondered what the outcome would be, and were suspicious of malignancy. I never had seen anything like it before. The end result is very favorable.

Dr. Emil Mayer, New York City: These cases are especially interesting. One particularly important thing is that the trained eye of the laryngologist sees at once that the diagnosis that is so often made in nearly every one of those cases is wrong, and that the cases are not of a malignant nature. I can picture for myself the view that Dr. Richardson had of that patient, with a proper light, where he at once concluded that the original diagnosis of malignant disease which had been made was wrong. I believe that we can in most instances make a diagnosis microscopically.

I must congratulate Dr. Richardson on his courage in putting in a knife to see if it was a bleeding tumor, and also on his successful removal of it intralaryngeally. The growth itself differs from most of the others on record, in that it was partialy external and partially within the larynx. It is very remarkable that a patient with so large a growth should be able to get along without a great deal of dyspnea. To my mind a case of this kind would be ideal for suspension. With very little effort one could draw the growth into a snare and remove it, and if necessary cauterize the place. In the case I reported a year ago the operation was done externally, and the growth was removed and the mucous membrane sewed over. I am confident that that was the only way the case could have been treated, for the reason that it was below the false cord and not above it.

Dr. Henry L. Swain, New Haven: Some of those present may recall a case in which I removed a tumor in the same manner as Dr. Richardson did, the report of which appeared in the Transactions for 1892. This growth, however, was lower down than it was in the case of Dr. Richardson, and it still holds the record for size in strictly intrinsic growths of the larynx. The growth was in the anterior commissure, and began only slightly above the place where the two cords come together, so that more of the base of the tumor was below than above. When the patient came into my office one could see nothing but the growth upon expiration, but during inspiration this tumor would move down so that the air could get by it. The patient had been living in that way with a very hoarse voice and with difficulty in breathing for nearly two months. He ran for a trolley car and fell down and was nearly asphyxiated, and relating that to me as I looked down into his larynx, I trembled at the idea of getting the tumor out. I had no idea of the up and down dimensions of the tumor; the diameter as I looked down upon it was just what would fill the chink of the glottis, but the vertical dimensions were three times the horizontal dimensions. I told the man that if he was a good soldier I would try to take it out. He consented and I put cocain in, and taking a snare got a good hold of the tumor twice. I tried to get the tumor out but failed. The man was getting blue in the face, but I put on a whole lot of power and got it out. I was more scared when it was finally out than I was before. It was a tremendous growth, much larger than was apparent to the eye, and still holds the record for size. I was prepared for the eventuality of hemorrhage, and told the man to throw himself forward with his head down, hanging over the end of the couch. He minded exactly as he was told, and hardly a drop of blood came away. He came back again on the third day and one could see then where it had been, just above the vocal cord. The patient had no recurrence. Dr. Richardson's specimen is a monster and the result of his skill is most gratifying.

DR. CHARLES W. RICHARDSON (closing the discussion): I wish to add just one word to Dr. Ingersoll's remarks, a point which he did not make quite clear. We were a little suspicious of the malignancy of this growth—in fact, we could not quite eliminate the matter from our minds—before we had the result of the pathologic findings.

Report of a Case of Carcinoma of the Larynx Treated With Radium.

BY ARTHUR W. WATSON, M. D.,

PHILADELPHIA.

A physician, about seventy-three years of age, came under my care November 24, 1914. There had been increasing hoarseness for a year, without inflammatory symptoms, pain or cough; the general health was good, the general history was negative.

Examination of the larynx showed a smooth, red, sessile growth or swelling on the left side beneath the vocal cord, extending from the anterior commissure backward about one-half the length of the cord, and downward from the cord about one-half inch. It seemed to involve the under surface of the cord. Movement of the anterior half of the cord was restricted, which caused bowing in phonation. The growth was sharply defined and the other parts of the larynx appeared to be healthy. A clinical diagnosis of carcinoma (epithelioma) was made. For obvious reasons a microscopic examination was not made.

When first seen the growth was one that could, undoubtedly, have been removed by laryngofissure, but in consideration of the age of the patient and the fact that he was in favor of trying the effect of X-ray or radium, it was decided not to operate.

The radium was applied to the outside of the larynx; eleven milligrams radium, filtration one millimeter of lead and one and one-half inches of gauze, for three hours. This was repeated in February (six treatments, 198 milligram hours). March 1st the radium was increased to twenty milligrams, filtration one milligram lead, one-half inch gauze, applied for three hours. This was repeated (six times, 360 Mg. hours). March 17th began forty milligrams radium, filtration same as before, applied for three hours. Repeated (six times, 720 Mg. hours).

Various applications were made, none of them satisfactory, and it had to be abandoned. The external application of the radium again instituted in the following dosage. Forty milligrams radium, filtration one millimeter lead and one-half inch gauze, applied five hours July and August (in all nineteen applications, 4200 Mg. hours).

September 23rd, about one month after discontinuing the radium, it was noted that the growth was apparently gone, left vocal cord a little slower in movement than the right, skin inflamed over larynx.

On the second of April, 1917, the patient returned with hoarseness, which he had noticed for a month or more. Examination showed a small nodule beneath the edge of the left vocal cord near the anterior commissure, the site of the old trouble. Radium was again used, a few days later, forty milligrams, for twelve hours. The larynx was examined two weeks later. The growth was found to be smaller, the neck inflamed. April 25th, only a slight thickening remained. The voice was again almost normal. The same condition was present when last examined.

From the results that I have seen in suitable cases, and with a better knowledge of the dosage, better results may be expected, especially if the radium can be applied from within the larynx, which would be made easier by a tracheotomy.

DISCUSSION.

Dr. Robert C. Myles, New York City: It is a question to be solved, whether in the early stages of incipient cancer it is best to immediately remove the cancerous mass with the adjacent tissues, and if it recurs to use radium, or whether it is best to use radium without microscopic examination. In this connection it would seem best to consider to what extent we would advocate either procedure if the growth was in ourselves. Judging from personal experience and observation, it is my belief that the average person who has a growth, or symptoms of a growth that is probably malignant, defers the question because they do not like to have it decided that they have one, and hope against hope that nothing will be found. There is unnecessary delay caused by that attitude of mind.

As far as the secondary or tertiary conditions of cancer are concerned, it seems to me to be unwise not to use radium for the hope it offers in the relief of pain and odor and the other phenomena connected with malignancy.

I think it was Dr. Mayer who made the remark earlier in the morning that we should first try to cure the patient and leave the scientific diagnosis unsettled. On the other hand, this is not conducive to progress; again, I do not know whether we can improve the physical conditions of a case which experience has shown us is malignant, by microscopic examination, nor do I see what the chances are of it being nonmalignant by resorting to such examination.

Dr. Henry L. Swain, New Haven: I am very glad that we have heard this paper, first because it was a model in the way it presented the dosage, method of treatment, length of treatment, etc., and secondly because it left us with a spirit of hopefulness.

This is again a case of the old, old story. We went through all this with the X-ray. We had in our hands a powerful means the possibilities of which we knew nothing, and we learned by exchanging experiences in the various meetings all over the country that X-ray burns were produced, that we must use a filter, that we had to have adequate dosage, if we were going to get results. We are now doing the selfsame thing with radium. In exchanging these experiences now, it seems as if we ought to come to some conclusions. I think one thing is clear—that we should not putter around with other means, and if we are going to use radium, we should get at it early. Inadequate dosage is worse than nothing. On the other hand, we must not use such a tremendous dosage that we either kill the patient or the surrounding tissues.

DR. E. FLETCHER INGALS, Chicago: I am under the impression that the dosage here is quite as important as in giving strychnin; that is, if you give too large a dose it will do a great deal more harm than if nothing had been given. If we read the reports of the men who have used radium, we will find that there has been in some cases great destruction of tissue from which some patients have finally died. Some patients can tolerate larger doses than others. It appears to me that radium, X-rays and sun's rays act practically in the same way: they burn in every direction where they can reach, unless properly screened. All the weaker tissues burn out first; but if the dose is a little too strong, the normal tissues will burn out at the same time. If this is correct, then the dosage is by far the most important thing; it must be worked out carefully. I tested each individual patient—a small dose at first, and repeated, according to its effects.

DR. ARTHUR W. WATSON, Philadelphia (closing the discussion): What I wished to bring out was the question of dosage. I am of the opinion that if the dosage is too heavy, so that the normal tissues around the growth are destroyed, cicatricial tissue is produced in which the malignant disease may more readily be reproduced. It seems to me that this may be the reason for failure to cure the disease in some cases. But I believe the best way of getting at the proper dose is by regarding what has been done in each case, instead of considering one application of radium the same as another.

Tuberculoma of the Tongue.

By Carl E. Munger, M. D.,

Waterbury, Connecticut.

Male, aged thirty years. The patient's tongue presented a crater-like cavity situated on the dorsom, at the junction of the middle and posterior thirds, in the median line, the prebase of the tongue. At the bottom of the crater was a marked ulceration covered with a yellowish slimy secretion, and from the central ulceration were narrow sharply marked ulcerations radiating to the circumference of the thickened and indurated mass. The surface of the swollen mass was yellowish in color, and there was marked and exquisite tenderness upon the slightest pressure, with much pain at every movement of the tongue. This pain was localized and not radiating when the tongue was at rest. There was present also the condition known as nigrities.

While the tubercular ulcer may be primary or secondary, a primary tubercular lesion of the tongue is extremely rare; although a few cases of tubercular infection of the tongue following traumatism have been reported. Speaking generally, the tongue is more often inoculated from the lungs than the lungs or larynx from the tongue.

The case reported presented a marked deviation from the usually reported situation of tubercular lesions on the tongue, which have almost invariably been stated to occur either on the tip or sides, this one being on the dorsum, a site on which we usually find a gumma.

The prognosis is not dependent upon the lesion itself, but is influenced alone by the fact as to whether the patient is suffering from concurrent lesions in other parts of the body which are not susceptible to improvement, or is he suffering from pulmonary tuberculosis, where there is a tendency to fibrosis? If the latter is the case, there is a fair degree of certainty that the tongue lesion may heal without local therapy, but if the pulmonary process is acute, with caseation and necrosis active, there is little chance of the tongue healing.

DISCUSSION.

Dr. Burt R. Shurly, Detroit: In a service of one hundred and fifty beds during the past six years in a tuberculosis hospital, we have had two cases of tuberculous ulcer of the tongue. Both of these occurred in a far advanced condition. I have yet to see, in my experience, a primary tuberculous larynx or a primary tuberculous ulcer of the tongue. Invariably, those cases have shown some other activity somewhere, if it is looked for sufficiently and thoroughly. These two cases suffered the most terrific pain and had progressed so far that there was absolutely no chance of relieving or curing the patients. We simply cauterized the ulcerations and made use of orthoform and other measures for relief, in the hope that they might be able to have a little more comfort and take some nourishment. In all these cases it seems to me that we ought to determine the relative frequency of bacilli. By this means the diagnosis is proved and syphilis is excluded. What seems to me marvelous is that we do not have some ulceration on the tongue, from the fact that the mouth in these advanced cases is absolutely alive with swarms of tubercle bacilli.

DR. THOMAS H. HALSTED, Syracuse: Seventeen years ago I removed the tonsils of a girl of twelve years, which were tuberculous. A year or two afterwards a surgeon removed the cervical glands of the neck, which were tuberculous. Two or three days later she developed a mastoid, and in the course of that developed a localized pachymeningitis. We thought it was tuberculous meningitis and expected her to die, but she recovered. Later on, in the course of two or three years, she developed tuberculosis of the phalanges, and one thumb and one finger were both operated upon by a general surgeon. She then developed tuberculosis of the nasal mucous membrane, which I curetted and applied lactic acid for a number of months. Then she developed a tuberculous ulceration of the alveolus, which must be now four or five years ago. Later, within the past two years, she has developed tuberculosis of the nasal septum, and the tuberculous process has extended so that now it involves the tip of the nose, and it looks as though she will lose the tip of her nose. This girl, during this whole period, has been a seamstress, and is of course living a fearful life, with a great deal of pain in the mouth and tongue, but she is very hopeful for some new means of a cure. She feels that ultimately she will get well. . In spite of all the disease processes, we have never been able to discover anything in the lungs. The diagnosis has been made through scrapings and the findings of the bacilli. The very interesting thing about this case is the long duration of the disease and the number of lesions which have developed in various parts of the body.

Dr. Harmon Smith, New York City: I do not think this was a tuberculous ulcer. It was in the median raphe, away from the border of the tongue, where irritation of a tooth would make an ulceration. It was a fertile field and one where tubercle bacilli would grow.

We should not confuse tuberculous fissures of the tongue and tuberculous ulcerations of the tongue with tuberculoma. It was about the location of the taste beakers in the median raphe.

Serial Frozen Sections of the Thorax From a Case of Aneurism of the Aortic Arch.

By George Fetterolf, M. D., and George W. Norris, M. D.,

Philadelphia.

The main points of interest in this report are as follows:

1. Photographs of the patient obtained at various periods during life showed successive steps in the growth of the protruding mass.
2. Frozen sections were made of a case which had had careful clinical study, and as a result, conditions as they had existed in vivo could be accurately reproduced and deliberately studied after death. Search of the literature reveals the fact that this is the first opportunity of this kind which has arisen and been taken advantage of.
3. The exact site of rupture, and the cause and mode of death, could be determined and depicted with a degree of accuracy, certainty and detail which would be impossible of attainment in an ordinary autopsy.
4. The hydrothorax, the pulmonary atelectasis, and the extreme dyspnea are explained in a manner which by any other method of study would have been much less satisfactory, if at all possible.
5. The last statement is equally true of the anatomic relations. The altered position and general changes in the bron-

chial tree and other thoracic contents are depicted in such a graphic manner as to show to the laryngologist and internist alike what does take place in the presence of such a lesion as this large aneurism. It also suggests what may take place in some of our future clinical cases in the presence of other disturbers of the normal intrathoracic relations.

DISCUSSION.

Dr. Greenfield Sluder, St. Louis: The labor in presenting Dr. Fetterolf's material is exceedingly great. We are constantly dealing with disturbances that seem trivial, which, if we bear in mind the relations within the chest, are frequently separated and put into a more serious category. It has been my experience within the past three months to, as it were, pick up two aneurisms of the aorta by virtue of dislocation of the windpipe, recognized in the laryngeal mirror, and a number of times I have outlined the same dislocations within the mediastinum, depressions of the windpipe, as a means of recognizing mediastinitis. Sometimes they are not of tubercular origin; more frequently I believe it to be tubercular. In one case that proved exceeding desperate, the infection was a primary tracheitis, going on for a length of time with mediastinitis and depression of the windpipe.

Stereoscopic Roentgenograms of the Head.

By J. M. Ingersoll, M. D.,

Cleveland.

The longer we have used the stereoroentgenograms, the more certainly have we been convinced of their practical value, for they give us definite information in regard to the nose and the nasal accessory sinuses, the brain and many of its blood vessels, the ear and the mastoid, which cannot be obtained in any other way. The size and boundaries of the maxillary and frontal sinuses can be distinctly seen. If there are any septa, tumors or foreign bodies in these cavities, they can be accurately located and defined. The ethmoidal and sphenoidal sinuses overlie each other, and are thus somewhat masked, but their position, relative to each other and the orbit and the other surrounding structures, can be clearly distinguished.

Skill in interpreting the roentgenograms can be more easily acquired by the surgeon than by the roentgenologist, for the surgeon has the decided advantage of being able to verify and correct the findings in the roentgenogram while he is operating. If the surgeon will carefully study the stereoscopic pictures before operating, compare his interpretation of the picture with the condition which he finds in the operation, and then study the picture again after the operation, he will soon acquire great skill in interpreting the stereoscopic plates.

In studying stereoscopic plates they should be examined from both sides. First, put the plates in the stereoscope with the film sides toward the mirrors. This will show the structures as we see them in the operation, with the external parts in the foreground and the deeper structures in the background. Then by reversing the plates in the stereoscope, turning the smooth sides of the plates toward the mirrors, the structures will be seen from the inside of the skull.

The technic for making stereoscopic anteroposterior radiographs through the frontal sinuses is described, as also the technic for making stereoroentgenograms of both mastoids on a single pair of plates.

These plates, when developed, are placed in the stereoscope, with the one having the right eye images in the right light box of the stereoscope, and the one having the left eye images in the left light box. With the glass side of the plates turned toward the mirrors of the stereoscope we view the mastoids from the inside of the skull, while with the film side of the plates placed toward the mirrors we view the mastoids from the outside of the skull.

DISCUSSION.

Dr. Robert C. Myles, New York City: Dr. Ingersoll has demonstrated it is much better than most of us believe it to be, and the topographic anatomy is shown better by this method than by any I know.

Dr. Hanau W. Loeb, St. Louis: It is really amazing how much more one gets to know about his work by studying one after another of the stereoscopic plates in every case which shows a disposition toward suppuration in any of the sinuses. It makes a man much better, even if he does not find in any particular case any essential value in the exposure. Taking the whole thing together, he develops an understanding of the subject far beyond what can be obtained by any other method.

The beauty of this work is that you can look at a case from before backward, or behind forward, by simply turning the plate around.

DR. LEWIS A. COFFIN, New York City: I just want to say in regard to the stereoscopic picture that there is no question but that more interest and more knowledge is to be gained from it. We must remember, however, that a doctor often sends a patient in from twenty, forty or fifty miles, and unless he can come down where we have such an apparatus he cannot see the results. There are times, therefore, when he must put up with the other picture, which is more or less helpful. We cannot always have the stereoscopic roentgenograms.

Lantern Slides Showing Normal and Diseased Sinuses of Children From One to Fifteen Years of Age.

BY LEWIS A. COFFIN, M. D.,

NEW YORK.

Dr. Lewis A. Coffin showed a double series of slides from X-ray plates of children's heads from one to fifteen years of age, one series showing normal and the other diseased sinuses. He said that one of the interesting things connected with the getting of the plates was the fact that nearly all the plates showing disease had been obtained from X-raying the heads of children from an eye ward of the Manhattan Eye, Ear and Throat Hospital without selection, that practically any case in that ward showed sinus disease, and he believed that this was strong presumptive evidence of the dependence of many eye conditions on the diseased accessory sinuses.

He called attention to the frequent involvement of the antrum, and stated that in this condition he saw the frequent cause of atrophic rhinitis and practically the universal cause of true ozena.

DISCUSSION.

Dr. James E. Logan, Kansas City: There is no question in my mind but that sinuitis results in atrophy—that is, the cases are usually preexisting sinuitis cases.

Dr. Robert C. Myles, New York City: I have never been able to find anyone who had seen a case which occurred after puberty—that is, anyone in authority. It is essentially a disease of child life. How much of it is retarded development and how much atrophic is another question. The most serious question is, how should we operate on the sinuses of these children; what is the best procedure? That can be developed in the future, I think. In regard to patients who are older, it has been my experience that at any age I get my most brilliant results, with regard to odor and continuance of discharge, by opening the antrum.

Dr. J. M. Ingersoll, Cleveland: I simply wish to ask Dr. Coffin whether these patients were operated upon endonasally or through the canine fossa, and what results were obtained.

It seems to me that we should not consider here the question of atrophic rhinitis. If this is to be a discussion on the origin of atrophic rhinitis, may I ask how it is that the orbital fissure from the anterior half atrophies also in the process? There is no sinus opening into that district.

Dr. Henry L. Swain, New Haven: Dr. Coffin has come to us with the most astounding assertions. I understand he has come with the proposition stating that he thinks he has further evidence of the origin of atrophic rhinitis in children. Then he comes forward with the statement that he thinks he can cure odor by opening up the antrum, and in his remarks he presupposes that these diseased antra began in childhood, produces atrophic rhinitis, and continues through years of life. Then he states he cures atrophic rhinitis of at least the odor by opening up the antra. I think those are rather bald statements and would like to hear them commented upon. Personally I have examined his statements and believe he is right.

Dr. J. L. Goodale, Boston: Dr. Coffin will have an opportunity for further elaboration of that point. It is in order for any member to ask questions regarding further information from Dr. Coffin.

DR. HANAU W. LOEB, St. Louis: This socalled purulent rhinitis which was described so well twenty-five years ago or so is exceedingly common, and from the fact that the nasal mucosa is affected, it is easy to understand that any of the sinuses more directly connected with the nose in childhood than in adult life should be similarly affected. If the mucosa of the antra is thickened in an existing case of suppurative rhinitis, it is very natural that it should show on the screen. That explains, to my mind, the reason that in so many of these cases Dr. Coffin was able to find evidence of obscuration of the antral field. However, this is an exceedingly common condition, and as atrophic rhinitis is fairly uncommon, he should explain why it is that so many get well without the production of atrophic rhinitis.

DR. BURT R. SHURLY, Detroit: I should like to ask Dr. Coffin how extensive is the operative procedure following this particular line of invasion.

DR. NORVAL PIERCE, Chicago: I am sure that some of these pictures are misleading. Last winter I had two cases—children ranging from four to six years—where the roentgenologic picture, according to the readings of the roentgenologist and myself, showed anterior disease—in one case bilateral, and in the other case lateral. I washed out three of these antra (in one case I washed it twice), and nothing whatever was washed out. They were quite dry antra. There is no doubt but what they were dry. In the past I have had a similar experience to that, where the discharge from the nose led me to have an X-ray taken, and where the roentgenologist said that the sinuses were undoubtedly diseased, but which were found healthy. So that we must check up these cases of supposed disease of the sinuses by other means than the X-ray.

DR. ROBERT C. LYNCH, New Orleans: Down South we see a number of cases of atrophic rhinitis, and I have two children of the same family, twins, two and a half years old, perfectly well developed cases of atrophic rhinitis. I wonder whether they have lived long enough to go through the period of inflammatory changes to give them atrophy. I do not think they are due to simple sinus disease.

DR. J. M. INGERSOLL, Ceveland (closing the discussion): We get new ideas in regard to the anatomic relations from the stereoscopic pictures, because we get the three dimensions. In regard to the use of stereoscopic radiographs, I think that unless a man uses them long enough to become familiar with them, and develops some skill in interpreting them, they are liable to stop using the method. If they do study them and check up the findings, I am sure they will become convinced of their value.

Dr. Loeb spoke of the value of reversing the plate. One of the great advantages of the stereoscopic plate is that you can look at it from both sides—that is, you see the structures in the operative field, and also from the inside, which you cannot see in any other way.

Shadows over the accessory cavities in the X-ray, whether stereoscopic or other plates, make one suspicious of involvement of the sinuses of the nose. I regard them simply as one of the confirmatory evidences. They are not positive. There are other things which enter into the causation of the shadows. A sinus which has thickened mucous membrane casts a darker shadow than one with a normal mucous membrane. The variations of the bone structures themselves, or part of the other bones beyond coming into the field, make one sinus appear darker than another. The bones do vary, so that a dark sinus alone, as seen in the plate, should make one only suspicious of that cavity.

Dr. Lewis A. Coffin, New York City (closing the discussion): Every case of atrophic rhinitis generally brings out something like adenoids and tonsils. Inasmuch as this is so, and that Dr. Loeb stated that it is not a very common or rare disease, I will convince him at least that it is as common as this. If you go in the hospital ward you will find almost all the children show disease of this kind. This does not mean that if you will take them off the street they will show the same percentage. They are simply diseased children.

I had a little patient in the same ward four or five years ago, and she is now ten or eleven years old. She had a keratitis, and had been in the hospital a year. We took out one tooth, both the milk tooth and the one above it, and cleaned up the antrum, and inside of a short time her eyes were open and they have been open ever since. I saw the child and examined her jaw, and one would not know she had even lost a tooth unless you counted the teeth on the two sides.

Somebody asked the question as to whether these all went on to atrophic rhinitis. I presume not. Some get well; but all the cases shown here are chronic cases.

There are changes in the lining membranes of sinuses that the microscope and pathologists cannot tell us. There are latent sinuitis cases in which the X-ray does not show much. I have operated upon a case and found no free pus and no pockets of pus, but every bit of tissue taken away the pathologist will tell you is just covered with disease. I think it is possible to get some of our worst conditions from latent sinuitis cases. The antrum frequently shows dark under the X-ray; you cannot even get water through, and yet there is no pus. I do not know what the degeneration of that membrane is, but it is full of polyps.

In regard to opening up the antrum and getting rid of the odor, because it is just as easy to do this in atrophic cases, go home and try it.

We have had one case in which the odor was as bad as you can think of, and the patient had never been treated for ozena, and nothing had been done but the antrum opened. In some of the cases we purposely left the ethmoids to pustulate and scab, in order to see the scabs without the odor, but the antrum is clear.

The Intranasal Drainage of the Frontal Sinus.

By E. Fletcher Ingals, M. D.,

Chicago.

The writer believes that in a large majority of cases of chronic frontal sinus disease, free drainage is all that is necessary, and that vigorous curettage is a bad practice, although gentle removal of polypi or granulation tissue with a curette, if present, would undoubtedly hasten the cure.

The furor for extensive mutilating operation on the frontal sinus has had its day, and now, more conservative intranasal drainage is a practice that is generally accepted.

The writer's attention was first called to the benefit obtained from intranasal treatment in 1893, when he succeeded in curing two cases by intranasal treatment, as neither of them would consent to an external operation.

As drainage into the nose was a prerequisite for treatment, the writer devised a burr with a safety guide to be run into the nasofrontal duct so that the duct could be enlarged permanently.

This burr made a canal six millimeters in diameter, which, however, had a tendency to contract. To prevent this latter, a metal tube was inserted into the canal. This metal tube was superseded by the spring-gold self-retaining tube now in use. This may be worn for a number of years and permits free irrigation by the patient.

The writer is convinced that the operation can readily be performed by any properly equipped laryngologist, and has placed the percentage of recovery of suitable cases as high as ninety-five per cent.

The instruments used are described, as also a detailed account of the manner of operating that the speaker has devised.

Further Report on the Intranasal Treatment of Accessory Sinus Diseases.

By Robert Cunningham Myles, M. D.,

New York.

In the past twenty-four years the writer has had much to say concerning the intranasal treatment of accessory sinus diseases. On the fourth Wednesday in January, 1893, twenty-four years ago, he read a paper entitled the "Diagnosis of the Diseases of the Accessory Sinuses and Their Treatment."

On account of the failure to secure uniformly good results, in antral suppuration, he made use of the malar ridge and canine fossa route; and then, after due consideration had been given to the cases, with more or less unsatisfactory results, it was noted that very favorable conditions nearly always followed when the window through the inferior nasomeatal wall was not less than fifteen millimeters in diameter, and never contracted to less than about ten millimeters.

The writer continues to prefer the nasal route to the antral one for operation on the sphenoidal cells, and strongly advocates making large openings in the anterior walls.

Partial success in an attempted submucous and subperiosteal operation on the anterior wall previous to removal of the bony wall, and the utilization of the membranes for covering the lower sections of severed bone, encourages the belief that if this procedure is well carried out it will aid materially in preventing the too frequent closure of the sphenoidal openings.

In the ethmoidal cases the classical operations of removal of the middle turbinal, the floors and the median walls of the cells are carried out.

The progress that has been made in the intranasal treatment of the frontal sinus diseases centers around the methods for making permanent large openings from the nose into the sinuses.

After removal of the anterior end of the middle turbinal and the anterior ethmoidal cells, the writer uses improved designs of his outward cutting chisels for removing as much as is possible of that part of the floor of the sinus which is formed by the nasal process of the superior maxillary bone. These instruments are used with a feeling of safety, as they automatically protect the dangerous areas in this region. Several of the most useful models are presented with this paper for your inspection.

DISCUSSION.

Dr. Emil Mayer, New York City: I would like to make an apology for the abstract editor, and would like to say that what Dr. Ingals complains of was not any garbling of any original paper, but a misstatement on the part of the stenographer of the discussion which was very voluminous on the symposium. The original statement of the stenographer as to Dr. Ingals' standpoint was quite in accord with what he says today. The trouble lay in an attempt, within one hundred and fifty words, to make a description of an operation which omitted some quite vital and important things. I will say this, that I question whether any laryngologist would take an abstracted report in any journal and would attempt to follow any method or description of any operation. He would surely go to the writer of the paper or to the Transactions of the Association, so as to get the correct report. However, all bad things, and this certainly was bad, as Dr. Ingals and I and the rest of you also will agree, have their good ends, and the good end lies in that we have again the pleasure of hearing from Dr. Ingals just how this intranasal operation is done, and how, after ten years, he finds that the operation is still one that he can recommend to us. So, if you will forgive the abstract editor in part and the stenographer in whole, I think you will agree with me that something has nevertheless been gained.

Dr. Joseph H. Bryan, Washington: I have been able to open up the frontal sinus in the living subject, too, but not in all cases. After having gone over the anatomic relations in the cadaver so many times, I have found out that a great many irregularities exist in the accessory sinuses, particularly in regard to the frontal in relation to the ethmoid, and it makes me hesitate when I hear this procedure so ardently recommended as it has been by Dr. Ingals. There must be some difference in the cases that we have been having. I am quite sure that no such method as the doctor recommends can possibly cure such cases as I have had. Now, I have never yet operated on an acute catarrhal or an acute suppurative inflammation of the frontal sinus. I have had considerable experience in this line. All of my cases have been of a severe form of chronic inflammation, lasting a number of years. After going over his papers time and time again, and trying to bring myself to believe that I could follow out the same line of procedure, I have been timid and have gone to the external method, and whenever I have one of these cases I have come to the conclusion that such an operation is not possible to relieve such cases. Now, is it possible in a frontal sinus, with a prolongation extending far into the frontal bone and extending back into the floor of the orbit and into the external angle, by more drainage to cure a case of that kind? Now, in these chronic cases there is nearly always caries of the frontal region, and it is not possible to cut the bone out or cure the condition by simple drainage, which this method is directed to, as I understand it.

In his hands, undoubtedly, the entering of the frontal sinus is practically an easy method, but is it a safe method or an easy method for the average rhinologist? While sometimes it may be very simple for those who are practiced in doing this kind of work, nevertheless, a man must always bear in mind that he cannot know what there is inside of that sinus without doing the external operation.

DR. HENRY L. SWAIN, New Haven: There are a great many cases where we refuse or hesitate to make an external operation. We are not all as successful as Dr. Bryan in having our operations clean up. Certainly, I think there is a vast field for the procedures of Dr. Ingals in the medium cases, and in the severe cases we will bow to Dr. Bryan. I have had some severe cases, but a lot of cases we could get well by the internal method. This is safe and sane. Dr. Ingals has proposed one to us. I must say I have not done it. I am like Dr. Bryan, afraid to do it, from my knowledge of it from his description alone, even though it is profusely illustrated. I seem to get along with milder means than even Dr. Ingals suggests, but I do get the cases well, and do not have to do the external operation only very rarely.

I have had this winter three cases which anybody would suppose ought to require external operation, where there have been spontaneous external perforations into the orbital cavity, and one where the disease went through the front plate of the frontal sinus on to the forehead. Presumably the process was severe enough to make the bone necrotic. All three got well simply by ordinary curettement of the frontal nasal duct and opening the abscess in the orbit. The bone of the external plate was not touched in any way at all. I have simply had good luck, but it shows the possibilities—that sometimes cases seem severe and yet do not require severe measures. There are three classes of cases—the ones that get well by simple curettement; those that require simple intranasal methods; and occasionally a case that requires the external method.

DR. HANAU W. LOEB, St. Louis: I just want to mention a procedure which Halle published in the past two years which makes all these external operations much easier, and that is to make a flap of the mucous membrane covering the superior maxilla anterior to the middle turbinate. It takes away that thickness of the wall and also permits much more ready access to the frontal sinus.

Dr. George E. Shambaugh, Chicago: The more I see of chronic accessory sinus infection, the more I become convinced that intranasal surgery is the proper treatment for the vast majority of the cases. Most of the cases can be cured by this method, and those which are not completely cured are with very few exceptions relieved of all symptoms which would justify an external operation.

In the case of the maxillary sinus I have found that by working through the nasal fontanelle in the middle meatus one can in a few seconds' time, with suitable instruments, secure an opening one-half by three-quarter inch long and one-half inch broad, which is ample for our purpose. This can be done with local anesthesia with much less shock to the individual than an opening in the inferior meatus. The old idea that an opening in the inferior meatus had an advantage because of its location near the floor of the sinus, does not hold true, thus securing ventilation of the sinus. No irrigation afterward is required.

The frontal sinus is considerably more difficult ofttimes to secure a sufficiently large intranasal opening to permit of free ventilation of the frontal sinus, and for this reason we are able less often by intranasal surgery to bring about a complete cure of chronic frontal sinuitis; but it is the rarest exception for us to find a frontal sinus infection where anything more than intranasal surgery is justified. It has been my belief for a long time that the external operation for the frontal sinus should be a tabooed operation except in the rare cases where, in spite of painstaking, careful intracranial surgery, severe symptoms persist, indicating retention and pressure and threatening intracranial complications.

Dr. Greenfield Sluder, St. Louis: I think that when Dr. Bryan takes the stand that there are cases with a ramification of the frontal sinus under the roof of the orbit, out into the external cochlear process of the frontal bone, when it runs back to the lesser wings of the sphenoid and extending more or less swollen beyond with granulation tissue, that case will in all probability not get on by simple drainage. Dr. Roe, if I remember correctly, in 1890, stated that he had deliberately left the antrum in one case filled with granulation tissue, prevented internal drain and left the granulation tissue alone, and that the case healed. Some five or six years ago I was in a frontal sinus externally and found it not specifically enlarged or ramified, but filled with granulation tissue. The case had been operated by someone else before, and the nasofrontal outlet was blocked in solid bone. There was great headache, and the bone was so healed that it could not be handled from within, so I went into it externally and found the cavity filled with granulation tissue, but put a drain into the nose and that was satisfactory. I left the cavity filled with granulation tissue and that case healed.

I believe that the value of a satisfactory drain for all of these, whether they be frontal, ethmoidal, antral, or sphenoidal, cannot be possibly too greatly exaggerated, but there must still remain a case that requires Dr. Bryan's procedure, that must of necessity justify his ideas concerning these cases. It is difficult to tell how far it really can go. In 1890 I had an antrum that had been suppurating four to five years. The operation that was done was the old Cowper through the alveolar. I kept it open with a plug, I washed it, and in some six months it stopped suppurating. It was always reinfected by coryza and healed with a few washings. The old gentleman died a few years ago. After I learned the intranasal procedure, I offered him that and explained that it was a better procedure and would relieve him of his trouble and his plug, but he declined to accept it.

Dr. E. Fletcher Ingals, Chicago (closing the discussion): Dr. Bryan thinks that if one cannot get into the frontal sinus. it cannot be operated on. This is quite right. However, I have been able to pass the probe into the sinus in ninety-five per cent of all chronic cases, but in about ten per cent I was not able to get it in the whole distance at first; then I ran a burr up perhaps three-eights of an inch, after which the probe passed easily into the sinus and the operation was completed. You notice that these probes are of different sizes and curves. If a probe will not go in with one curve, make another one; if a large one, one and one-half millimeters, will not go in, take one that is one millimeter in diameter. If one can do the operation, possibly in five per cent of the cases an external operation may be desirable later on. I would not claim this as a cure-all by any means. The question as to whether drainage is sufficient to cure these cases or not is one that we have to determine by experience. Experience seems to show that it is sufficient in most cases, even if there are granulations. It seems to me that scraping out all of the mucous membrane in the sinus is absolutely bad practice.

I do not recall any chronic case in which I have attempted to probe the frontal sinus in which I have not succeeded—at least, after I have run the drill in for perhaps three-eighths of an inch; but I will admit that it has occasionally taken me as much as an hour to do it. But if I can get the probe in once, the next time that I put it in I can make a large opening very easily.

I believe that practically all of the cases will heal if we give them drainage, and I think that most of you will believe this.

Dr. Robert C. Myles, New York City (closing the discussion): If you are going to obliterate, you have to obliterate completely; but if you leave one small piece of mucous membrane in the sinus, you will have trouble. Patients are coming to me all the time from the general surgeons with imperfect drainage. If you notice, in my paper I mentioned the fact that they opened the sinuses and left them with a socalled polypoid condition; they simply packed them. The reason of that is that these are not granulations, but an edematous condition called granulations. They will disappear; the condition is the same as the watery eye or the postorbital eye. It is a circulatory condition and not a pathologic one. I admit, of course, that granulation sometimes comes from diseased periosteum.

But fundamentally, the crucial point in all of these cases is to get it open and maintain it open and keep it open; and the results in this way are better so far. I believe that five or ten per cent of the cases have come to external operation, but we are now able to cure a large percentage of them by the internal method. The question of the necessity of distinction enters. I have lots of cases which heretofore I would have determined to require the external operation which are now operated by the internal procedure.

Vincent's Angina.

By Thomas Hubbard, M. D.,

Toledo.

It is worthy of remark that many of the clinical articles are reports of very serious or fatal cases. This group of fatalities, in a disease that is usually transient and curable, and taking into consideration the rather scanty literature, arouses the suspicions that a large number of cases are not diagnosed. Laboratory study of all ulcers and false membranes is necessary to positive, timely diagnosis.

He calls attention to perborate of sodium as an efficacious local medicament. The nascent hydrogen dioxid penetrates and disintegrates necrotic tissue and would be, theoretically, an ideal inhibitor of anaerobic bacterial growth. Perborate of sodium is a constituent of the foaming dental powders, and has stood this empirical test with credit.

The local treatment resolves itself into careful applications limited to the diseased tissue. The glycerole of iodin (iodid of zinc, 2; idodin, 3; aq. dist., 5; glyc., 10) applied to the tonsil crypts, is probably the most efficacious antiseptic.

Theoretically the arsenical group is specific in action, and the writer thinks that experience is proving favorable.

He reports a case which presented such a hopeless condition prior to the administration of neosalvarsan, and the improvement following was comparable only to that which we see in diphtheria cases following a proper dose of antitoxin.

In cases that do not yield promptly to this treatment cacodylate of sodium, two and one-half to five grains, repeated in twenty-four or forty-eight hours (if kidneys are not affected). In the malignant type, not improving under the above treatments, give neosalvarsan 0.6 gram (average body weight adult) intravenously.

DISCUSSION.

Dr. Emil Mayer, New York City: I would take some exception to the originality of the use of glycerin of iodin. When I first called attention to this affection we know so well now, I then spoke of the use of idoin, iodid of potassium, and glycerin as a local application. I have since learned to know the tremendously valuable effect of the salve, either injected intravenously or applied locally.

A very short while ago a patient presented himself at the Mt. Sinai Hospital, New York City, with a very large ulceration at the under surface of the tongue that had been existing for a couple of months and due to the bacillus Vincent. A local application of the salvarsan with an intravenous injection, the Wassermann having first been proven negative, resulted in a cure within forty-eight hours. I would like, however, to see some of the results from the application of the salvarsan locally, without the intravenous injection. It is very possible, indeed, that the application itself would do all that we would require, and the patient might not even require an injection; but it does not mean by any means that because a patient has recovered that that patient has had syphilis, but that this arsenical preparation has a beneficial effect on the treatment and cure of this disease.

Dr. Thomas Halsted, Syracuse: About a year ago a physician consulted me with the statement that for six weeks he had had a severe tonsillitis. He had contracted this, apparently, from dancing one evening with a school teacher, who had at the time a sore throat which was diagnosed the next day as diphtheria. She died in the course of ten days from Vincent's angina. This doctor developed within a short time an ulcerative sore throat. He was seen by a number of specialists, but the diagnosis was not made, and he finally came to me. I found an extensive and deep ulceration of both tonsils and a marked cervical adenitis. The ulceration was soon proven to be Vincent's angina microscopically. A short time before this my attention had been called by a general practitioner to the possible use of enesol in the treatment of Vincent's angina. Enesol is a French preparation of arsenate of mercury. We used enesol hypodermically and did nothing else. There was no local treatment whatever. The improvement was marked, and at the end of six days, during which time three treatments were given, the ulceration was perfectly healed. Since this case I have treated not less than six or eight cases of Vincent's angina with enesol, and all of them with most satisfactory and rapid improvement. I think enesol is a specific for

Vincent's angina, and preferable to salvarsan, because of the greater safety and simplicity of the treatment.

Dr. Thomas Hubbard, Toledo: I am very glad that Dr. Mayer mentioned the use of iodin. I would be interested to know the formula he used.

This formula is iodid of zinc, iodin, glycerin. Whether it is more penetrating than the ordinary iodin glycerin preparations, I do not know.

As to the choice of the arsenical preparations, I think that is a question of clinical proof, and I think Dr. Halsted's report only goes to show that the use of some member of the arsenical group is a dependable treatment of Vincent's angina type of infection.

General Streptococcic Infection Through the Accessory Sinuses and the Tonsil.

By T. H. Halsted, M. D.,

Syracuse.

Chronic suppurations in the sinuses are by no means local diseases in the sense that their effects are confined to these organs.

It is the chronic infections, the staphylococcic, pneumococcic and streptococcic, that are perhaps of greatest importance, because of the insidious and slow degeneration which their absorption produces in the whole system. It is these which are so commonly overlooked and their true significance so little appreciated by both specialists and the general practitioner.

Of equal and perhaps greater importance than local infections of the nose and their adnexa, though only in degree, are the infections of the teeth and of the tonsils.

The tonsils, whether the faucial, lingual or nasopharyngeal, in a normal healthy condition, undoubtedly offer a hindrance and are a barrier to the farther ingress of pyogenic organisms to the lymph or blood stream. Conversely in a diseased and dilapidated condition, these same structures offer a habitation and afford the easiest way, becoming the open portal, for the passage beyond of the invading army of microorganisms.

A mixed infection of the tubercle bacillus and the streptococcus is of frequent occurrence. Just as tuberculosis is conveyed from one individual to another through the inhaled sputum, and is mildly contagious, so probably is rheumatism—i. e., infectious streptococcic rheumatism.

It is a matter of constant clinical observation that with enlarged and diseased tonsils, not regarded as tubercular, enlarged cervical glands, thought to be tubercular, are often associated. The removal of such tonsils is usually followed by a reduction in the size of the enlarged glands.

Endocarditis and arthritis often supervene or follow an attack of acute tonsillitis.

Chorea is now known to be a rheumatic or streptococcic infection of the nervous system, that the improvement results because of the removal of the primary focus of infection, the tonsil and the adenoid.

In childhood, tonsillar and lymphoid tissue is most prone to acute infection, and it is at this time of life that acute inflammatory rheumatism and endocarditis, usually following an acute tonsillitis, is most likely to occur.

We shall soon be classifying a great many affections now regarded and treated as separate entities under the general classification of streptococcic disease.

The streptococcus is transmitted from one individual to another through direct or nearly direct contact, such as in the act of coughing, sneezing, kissing, the organism being inhaled into the mouth with the mucus from the infected person, or it may enter through traumatism by means of an infected instrument, or again it may reach the mouth through food, particularly milk, the milk having been infected by those handling it.

As for the treatment of streptococcic infected tonsils, complete enucleation (tonsillectomy) is the rational procedure.

After the tonsils are removed the patient may still show evidence of remote or general streptococcic infection. This means simply that there still remains a focus somewhere, possibly in the adenoids. Possibly the tonsils were not completely removed, even though the operator was most careful, a small piece, no larger than a pea, remaining in the supratonsillar fossa or at the base, adjoining the lingual tonsil; and if such is the case, this fact should be recognized and dealt with by a second operation, the sooner the better. Again, an apical tooth abscess may have been overlooked or the difficulty may lie in a secondary focus in the gall bladder, the appendix, some joint, the endocardium, pleura or other localized area.

The Susceptibility to Infection Manifested by the Remains of Incompletely Removed Tonsils.

BY HANAU W. LOEB, M. D.,

ST. LOUIS.

If a portion of the tonsillar lymphoid tissue is left after operation, especially if it happens to contain a crypt, it is very much inclined to persist in statu quo. It may never occasion any unpleasant result, but it is present nevertheless with its susceptibility to infection, reduced though it may be. What is considered an atrophied tonsil usually signifies that the tonsil tissue has become somewhat more covered by the anterior pillar and has in part simply disappeared from view.

There must be a not inconsiderable number in which tonsillar stumps remain, even in the practice of the most experienced operators, and the writer presents five cases showing infection originating in such tonsillar remains.

These casese definitely show that small masses of tonsil tissue overlooked, or at least not removed at the operation, are susceptible of infection with remote effects similar to those which follow acute tonsil infections.

They must have their counterpart in the practice of other laryngologists, and from his own experience must be common enough to constitute a fairly definite clinical entity.

They present a decisive argument against any form of operation which does not contemplate the entire removal of the tonsil, especially where there has already been some infective processes originating in the tonsil.

They suggest the advisability of following up cases of tonsillectomy to determine whether any portion remains and whether it has become a focus of infection.

DISCUSSION OF PAPERS OF

DR. T. H. HALSTED AND DR. HANAU W. LOEB.

Dr. George B. Wood, Philadelphia: I desire to call especial attention to the difficulty in recognizing when a tonsil is responsible and when it is not responsible for a given infectious condition. According to my own experience, inspection of the tonsil itself in the majority of cases side little in this determination. Small innocent looking tonsils can give rise to severe general infections, while, on the other hand, large swollen tonsils with masses of epithelial débris in the crypts are frequently found in apparently normal individuals. Of much more value is the history of repeated attacks of tonsillitis, their relation to the general infection, and the presence of cervical adenopathy. The bacteriologic study of the contents of the crypts of the tonsil will often give important data. This study can be carried further by determining the relation of the bacteria obtained from the crypts to the blood reactions of the individual. In a person suffering from chronic tonsillar absorption for a considerable length of time, it is probable that a certain amount of immunity against these organisms has been established. If agglutination of the bacteria from the crypts or a complement fixation can be demonstrated, there would be good reason to believe that a certain amount of absorption had taken place through the tonsil. From a somewhat limited number of cases this reaction has been found in a certain number of chronic cases, though in acute tonsillar infections the immune bodies could not be demonstrated, probably because sufficient time had not elapsed for their formation. The importance of these reactions can only be determined by a large series of cases, preferably carried out by a number of observers.

Concerning the possibility of recurrence of tonsils after removal, I believe that a certain amount of actual recurrence can take place. In these cases the new tonsillar tissue is superficial and is found simply as small lymphoid follicles scattered over the surface of the scar tissue. More frequent and more serious apparent recurrences are simply hyperplasias of tonsillar tissue left behind at the operation.

DR. BURT R. SHURLY, Detroit: Many people think that when their tonsils are removed they will never have another sore throat, and that is not probably explained sometimes by the man who does the operation. Again, we have certain islands of tissue that are perhaps more frequently left in place, and undoubtedly, if we take into consideration all the different operations we have done throughout the years, we will find a very considerable number of incomplete operations. On the anterior pillar itself, very frequently even when we think we have accomplished a very complete operation, we find tiny islands of tissue which can only be observed by a most thorough examination. Many times when we think we have done a most complete operation and go back over the ground again, one, two or three times, we still find some tiny islands of tissue we have left; and if this happens to be on the anterior pillar, it frequently gives rise to more trouble. I do think, though, that there is great danger of exaggeration as to just how much damage results from leaving some of these tiny islands of tissue.

DR. THOMAS HUBBARD, Toledo: I have in mind a throat in which there was perfect enucleation of the tonsil, and yet the patient returned several times and said that she was having recurrent attacks resembling those which she previously had. Finally, one day she returned with a distinct swelling. I was able to pass a probe into the fistulous tract, which evidently led into the muscular tissue, and that was where the trouble was. There was no tonsillar tissue left at all, but showed a most thorough operation. It is probable that sometimes we leave one of the small fistulous tracts which have been part of the peritonsillar suppuration at some previous time. I would say that during these attacks she had a recurrence of the arthritic symptoms, just the same as when the attacks were due to tonsillar and peritonsillar inflammation; but it was a distinct fistulous tract leading into the deeper tissues, and I have not had an opportunity to attempt to eradicate that, although it would be comparatively easy.

DR. J. PAYSON CLARK, Boston: There is one point in Dr. Halsted's paper which I would like to emphasize, and that is the possible relation of unsuspected conditions of the teeth to general infection. I have been surprised at the number of cases in which a tooth giving no symptoms locally at all, was in very bad condition, and treatment of that tooth at once relieved the general symptoms.

I have been surprised once or twice, and several of the cases I have been able to explain, where I was perfectly positive I removed the tonsil, capsule and all, where the capsule was perfectly smooth and the fossa quite empty after operation, and a year or more afterwards there has been an appearance of tonsil on one side or on both sides. My explanation was that some of the lymphoid tissue of Waldeyer's ring in the healing process has been drawn up into the region of the tonsil. That tissue drawn up differs from ordinary tonsillar tissue in that no crypts appear in it. I have never seen any inflammatory process.

Of seventy-three cases operated at the Massachusetts General Hospital before we had begun to do the capsule operation, in sixty cases the tonsil tissue was still visible; twenty of the cases gave a history of one or two attacks of sore throat since the operation. Of those attacks only ten had been definitely determined upon to be tonsillitis; at least two-thirds of the cases of tonsillotomy had had no subsequent trouble. There were twelve cases of illness since the operation, but none of the cases of illness were of the kind to be attributed to any infection from the remains of tonsillar tissue; these cases were all children.

Dr. Henry L. Swain, New Haven: The fact that we do not have more knowledge of the return of tissue after enucleation, I believe is due to the fact that in lots of our cases the person is not as susceptible as are those from whom we do hear. Certain cases show marked susceptibility to inflammatory seizures. If you leave the slightest bit of tissue, you get a report of sore throat and constitutional symptoms, etc. In other cases you can leave three times as much tissue and there is no report of any trouble whatever.

In my home town, a year ago, we had an epidemic of streptococcic sore throat, due to milk infection. In all we had one hundred and twenty cases. I took a great deal of interest in looking into the question of the presence or absence of tonsils. Out of that group there were between twenty and thirty cases who had had thorough removal of the tonsils—

absolute tonsillectomy. None of us would have been dissatisfied with the results of the operation had we done it ourselves. In these cases we sometimes got more trouble than in those in which there is an abundance of lymphoid tissue. The five cases which gave me the most trouble, with abscess formation, edema of the larynx, were in cases of complete tonsillectomy.

The probabilities of general infection such as Dr. Halsted speaks about is illustrated in a most interesting case I had recently, where the patient had a simple sore throat and then developed general poisoning of the system from the effects of inflammation. As a result of that he had a large abscess at the back of the neck which seemed to have a formation of membrane in it, so that one expected to find pseudodiphtheritic discharge. He was given autogenous vaccines in large doses, and it was well carried out in every particular. Six weeks after the original sore throat he came down with another sore throat in which he had exudate over four distinct strips of tissue, both tonsils and both lateral columns of the pharynx were covered by a false membrane. Cultures from this showed it to be streptococcus. He did not have a distinct streptococcemia.

DR. GREENFIELD SLUDER, St. Louis: It is an interesting question all the way through, and that is the matter of peritonsillar infiltrate. The aftermath of the case, the future course of the case frequently has been influenced by the infiltrate that was about the tonsil. That infiltrate seems to me to frequently take the part almost of the tonsil itself. I have seen them a few times, and latterly I have seen a few cases in which the infiltrate behaved as the tonsil—began with a sore throat and suppurated in both cases. The tonsil was perfectly enucleated and preserved in a bottle. I make it a practice in operating upon cases to save the tonsils from every individual, and later on I frequently find that I possess a number of tonsils and capsules which are perfect; but, as Dr. Clark has expressed it, the lymphoid tissue of the base of the tongue has grown up into the fossa and looks very much like a tonsil. The peritonsillar infiltrate may, furthermore, hypertrophy and later on look like tonsil, as if something had been left, unless very carefully inspected. With that end in view, some three months ago, in the presence of a very large infiltrate, I removed the tonsil with a guillotine and found something which looked like the tonsil still in the throat. I then took out an infiltrate, the size of the original tonsil, and the infiltrate on the opposite side remained. Subsequently sore throat developed, and developed in the infiltrate that was left. The side from which the infiltrate was removed escaped.

DR. HARMON SMITH, New York City: I believe that nature requires a certain amount of lymphoid tissue in that region, and, just as we see when we remove the soft palate for malignancy, the posterior tips of the turbinate hypertrophy to prevent regurgitation of food in the pharynx, so the remaining lymphoid tissue undertakes to carry on that element of protection which the tonsils previously did. I do not believe that spontaneously tissue resembling the original tissue will spring up. I know of instances in which, three or four times after the removal of the tonsils, from childhood on up to puberty, where each time every microscopic evidence had been removed, yet there still recurred certain islands of lymphoid tissue, and around the islands there was formed a certain kind of capsule.

If you remove tonsils and leave lymphoid tissue at the base of the tongue, in such infections as from milk, etc., the lymphoid tissue at the base of the tongue will produce tonsillitis just the same as the original tonsil.

DR. JOSEPH GOODALE, Boston: In certain children who may have tonsils and adenoids removed by operation we may have a condition of partially developed anaphylaxis. It is not a true vasomotor rhinitis, but a condition of snuffing, blocking of the nose, and the symptoms which appear are those of a tendency to taking cold. There may be possibly a portion of tonsils left, but what we should do is to look somewhat further than the throat. We should see if the child is taking more milk than it is accustomed to in the summer, or an extra cup of cocoa, or an extra egg; it seems to me, before we should proceed to a further operation on that throat that we should examine very carefully this question of diet. I would suggest that any of this unusual food should be diminished and restricted, and I am sure you will find that a certain percentage of these cases will lose their symptoms. They will freely disappear without another operation, or loss in weight, but a gain in weight.

Dr. Hanau W. Loeb, St. Louis (closing the discussion): I have spoken of the actual leaving of portions of the tonsil in some instances by myself, and in some instances by other operators. I am sure that if operators will examine their cases, one, two and three months after operation, they will find not infrequently that a small mass of tonsillar tissue remains. It so happens that in two instances I was able to verify the fact that I had left a portion of the tonsillar tissue there, because I had the tonsil in a bottle, and a careful examination of the capsule revealed where a very minute portion had been omitted.

With reference to the inclusion of some tonsil tissue in the scar, I feel, at least in one particular case I reported, that the inclusion was there before the scar; in other words, I left a mass there and the scar covered it over. This was the case in which there was a minute abscess in a little crypt. This patient had several attacks of appendicitis which went on to operation, and I feel the little piece I left there may have been responsible.

We should not hesitate to let our patients know when we have performed an incomplete operation. This is better than having an acute infection come on and someone else tell the patient. I do think we ought to protect ourselves in that respect. I for one never hesitate to tell the father or mother of a child that I have left a small portion of the tonsil. Of course, years ago that was fairly common, but I am thankful to say it is uncommon now.

I wanted to bring the matter up to the association, not as an argument for tonsillectomy, but for complete tonsillectomy, and also to assert that the mere statement that an operator is going to do a tonsillectomy does not signify that he is doing or has really done a complete tonsillectomy operation.

Presentation of Pathologic Specimen of Large Tumor of Pituitary Gland.

By Robert C. Lynch, M. D.,

New Orleans.

The man was a quadroon, about six feet one inch tall, with hands and feet somewhat larger than usually seen, though not sufficiently so to be suggestive. He was well developed to the point suggesting fat, with a fairly developed chest and mammary gland, and small hips, resembling in a degree the feminine type.

About the end of July, 1916, he had profuse nose bleed, following heavy work, with recurrences daily, when the nose began to block up, voice became hoarse and the eyes began to protrude. Upon examination the septum was deviated strongly to the left, the right nostril filled with an irregular spongy red mass which bled easily upon manipulation; pupils were dilated and did not respond to light or accommodation; pulse, eighty; eye grounds show slight pallor with double temporal hemianopia or loss of vision over the outer half of each eye. A specimen of tissue was removed and the laboratory reported back: "A sarcoma."

The patient in December had the same symptom complex apparent, only slightly aggravated. The exophthalmus was very marked, the superior maxillary prognathism was apparent. The mass in the nose was as described and could be seen in the nasopharynx.

An attempt to remove the mass through the nose only succeeded in getting out a small part. The tissue was reported as granulation or inflammatory, but not malignant. Upon the second trial a facial decortication was done, and at the same time the right antrum opened. This gave plenty of room to remove the mass more completely, but when the roof of the nose and over both orbits were found pulsating, and with the finger he could feel the meninges and see the dura, he was convinced that it was time to stop, fearing lest the sudden loss of support would produce a hernia.

The nose was packed, a large postnasal plug introduced, tying the attached string to the ear. He was returned to bed in good shape, but upon waking from the anesthesia he caught the string and pulled upon it with such force as to force the postnasal plug through the exposed dura and into the brain. He died from a basilar meningitis.

The specimen was presented for observation, together with the X-ray plates and a microphotograph of the tumor which is characteristic of pituitary gland.

Report of a Case of Abscess of the Frontal Lobe of the Brain.

By Robert C. Lynch, M. D.,

New Orleans.

Mrs. L. had been an invalid and a sufferer for considerably more than fifteen years, her condition varying only as to the degree of her suffering, which was often very great. The pain and headache from time to time confined her to bed for varying periods, and frequently the intensity was so great as to cause convulsions accompanied by a severe rigor and a bending backward, so that whiffs of chloroform were used to bring about relaxation. There were varying terms of slow recovery to a condition that enabled her to leave her bed, but she never was well, and had headaches which were almost continuous.

In 1907, following right sided abdominal pain, an ovary was removed with no improvement. In 1909, a panhysterectomy was performed; following this a period of relief ensued, but in 1912 the headaches grew so intense that a rhinologist was consulted. A right maxillary sinuitis was irrigated for nearly a year, but with only varying relief. A change was made, and the second colleague removed the right middle turbinate, opened the right frontal intranasally, and curetted the ethmoid cells. There followed a long period of treatment without relief.

Late in 1913 an acute exacerbation in the right frontal sinus occurred, demanding an external operation. There seemed to be no improvement afterward.

Three years later, after careful examination, Lynch found the right antrum to have a large opening through the inferior meatus, and irrigation of this cavity showed slight pus at times, but which bore no relation to the headaches.

The postethmoid region seemed full and bulging, and he was able to wash nearly a teaspoonful of nonoffensive pus from the sphenoid sinus. Treatments of this character seemed to be followed by considerable relief from headaches and relieved the chronic hawking and spitting complained of, though she had during the period of this treatment one of the chill-temperature headache attacks, which seemed to have a certain regularity as to recurrence, regardless of the treatment employed.

There ensued now a gradual decline in body strength and a slight dullness of mentality, with no desire for food and persistent headaches of increasing intensity, blood counts rising to 17,000 with 90 polymorphonuclears. Blood pressure, 110—rather low; no focal symptoms of any kind, as determined by a most searching examination.

The decline reached the state of involuntary micturition and defecation, mental dullness to almost coma, and inability to suck fluids through a straw, to be roused only on the greatest excitement, and speech then inaudible—still no focal symptoms.

On her return to the hospital, some months later, Lynch determined that the symptoms were those of brain abscess in the silent area—the frontal lobe; accordingly, he opened up the old frontal sinus wound, which was apparently perfectly healthy and sound, but to his surprise there existed a small cavity holding about fifteen drops of milky pus, looking much like an old mucocele. On removing the membrane from the posterior wall the bone looked normal and was intact completely. He could not feel satisfied, however, that the findings were sufficient explanation for the symptoms, and took down the posterior sinus wall. The meninges seemed normal and not adherent in any direction, and there was no bulging or undue pressure apparent. He split the dura, and the brain surface seemed normal; so he explored with aspirating syringe and large needle, applying suction as soon as the needle was below the surface. After about an inch deep, following the line of the base of the skull and away from the longitudinal sinus and under the anterior horn of the ventricle, there gushed into the syringe about three drams of yellow green pus. He withdrew the needle and prepared the bone opening and area for the reception of one of Mosher's wire gauze drains; reintroduced the needle and withdrew four drams of pus. Then he opened with a scalpel what he thought was an abscess cavity, but try as he might with needle, knife, or forceps, he could find no evidence of the abscess. Finally, he passed a small rubber tissue drain into the brain, in the direction and to the depth of which he had aspirated pus and dressed the wound. Upon recovery from the anesthetic the patient was conscious, and in six hours addressed him voluntarily, complaining of some soreness about the wound, but not of much headache.

The pus showed staphylococcus aureus in pure culture, but not active, growing feebly on culture.

DISCUSSION.

Dr. Thomas H. Halsted, Syracuse: In this connection I shall report the present condition of a case of hypophyseal cyst which was operated two years ago and reported to this society in 1915. It is the case of a little girl, eight years of age, who at that time had as the leading symptoms great increase in size of her head, great increase in weight, weakness, constant shaking of head with incoordination of movements, so that she was unable to feed herself; optic neuritis, sharp headaches, very striking change in color and texture of hair, polyuria, and disturbed mental acuity. The operation was done through the nose under local anesthesia; it was the modified Hirsch operation, and the growth proved to be a cyst of the hypophysis. The patient was much improved temporarily. Some months later the cyst recurred and a second operation was refused by the parents. It is now about three years since the original operation. I have not seen the child in a year, but saw the father within the past six months. He said that the child now has simply an aggravation of all the symptoms; she has become very fat and large with great weakness, and the blindness is nearly total.

Dr. Henry L. Swain, New Haven: I would like to ask Dr. Lynch what was the condition of the brain of this patient.

I had a case which I saw but once, in which there was bitemporal hemianopsia which was due to a tumor of the pineal gland, in which there were headaches somewhat similar to the second case.

Dr. Robert C. Lynch, New Orleans (closing the discussion): In reply to the question by Dr. Swain, as to what was the condition of the brain of this patient, I would say that the brain did not show anything pathologic at all, except that the coverings represented the usual findings of death from meningitis.

We scraped up in New Orleans three other cases of pituitary tumor, and all of them showed on X-ray a very marked enlargement and beginning protrusion backward of the sella turcica. They all showed the same phenomena as regards the eyebrows, and they all showed the symptoms of lack of function of the pituitary gland.

Three Bronchoscopic Cases of Dentist's Origin.

By Burt R. Shurly, M. D.,

Detroit.

As these three cases occurred during the last decade in a city of an average population during this time of 500,000 people, and as we have an estimated population of over 100,000,000, it might be fair to conclude that possibly six hundred cases of similar accident had occurred in the United States and Canada, perhaps, during a similar period. It might, therefore, be of importance to devise a special protective device to prevent the sudden inhalation of foreign bodies during dental procedure.

Again, the question of the medicolegal problem, with questions of responsibility as to whether these accidents involve ordinary care in dental procedure, might be of interest, although exceedingly delicate ground to tread upon, as one of these cases involved a very considerable law suit, and the other two were allowed to pass with nothing more than an attempt to collect the surgeon's fees from the dentist. The medicolegal possibilities did not come to actual trial.

The peculiar relationship of laryngology and clinical medicine and its great importance, one to the other, was again illustrated by the fact that two of these cases first came under my observation as referred cases for clinical examination of the throat and chest on account of long, persistent cough which simulated pulmonary tuberculosis.

Case 1.—Miss S. stated that her cough dated immediately after the extraction of tooth and gas anesthesia. Radiogram showed tooth. Trachea was not cocainized and bronchoscopy failed. Tracheotomy and subsequent bronchoscopy resulted in removal and cure.

Case 2.—Married woman. Cough followed nitrous oxid and tooth extraction. There was a marked odor of rubber. The air supply of lower part of lung was cut off, and piece of hard rubber, which had been a part of a dental mouth gag, was brought up to the trachea, slipped from forceps, but fortunately coughed up soon after by the patient.

Case 3.—Mr. L. W. B., age forty-nine years; architect by profession; was receiving treatment from a dentist preparatory to filling a tooth. The cavity was treated with a dental burr, which in this particular case was held between the thumb and forefinger during the process of application. This tiny instrument suddenly slipped and was inspirated into a lower division of the left bronchus. After this remained in the bronchus for ten days, the patient was sent from his home in the central part of the State of Michigan, to the Detroit Eye, Ear, Nose and Throat Hospital. The X-ray examination reported—anteroposterior and lateral plates were made of the chest: "We find a shadow in the lower left thoracic region, which we think should be interpreted as a metallic foreign body in the lower left bronchus." After a thorough cocainization with novocain of the respiratory passages, with cocainization advanced as the tube was introduced and followed to within the lowest terminations possible, and assisted by Dr. Hickey, I was able to extract this foreign body, which was pointing with the needle part upward. The patient recovered completely without symptoms.

DISCUSSION.

Dr. Thomas Hubbard, Toledo: It certainly seems that the position of the patient's head in the dental chair is very conducive to the loss of a foreign body in the throat or air passages. Fortunately, most of them go into the gastrointestinal tract. I have in mind a case which recently occurred of a nurse taking dental treatment, in which a small dental burr was lost.

She was taken violently ill and returned to the city with violent abdominal disturbances. The X-ray failed to locate the dental burr, but the operation was really appendectomy, the surgeon expecting to find the dental burr in the appendix. Had this girl, for instance, not had the surgical operation, the appendectomy, in all probability the whole thing would have been laid to the dental accident.

Another case comes to mind in which a tooth was lost and remained in for several years and was then spontaneously expelled. The patient was not very ill any of the time and finally expelled the tooth spontaneously.

The third case is that of a patient, a woman, who carried a fragment of Allen's dental cement, which is quite as firm as a tooth structure, in her left lung for seven months, and during that time had all the symptoms of advanced tuberculosis, with a very considerable abscess cavity around it. This brings up the question, as raised in this case, as to the legal complications which are liable to come up. In justice to the dental profession, I believe we should take them into our confidence in all cases of liability on the part of the dentist, and he should be taken in to see the operation, as occurred in this case. I am sure the dentist paid the bill—or rather he spoke as if he felt it to be his duty to do this. He was at the operation, and this relieves us of any possibility of unfair treatment towards the dentist—that is, if one takes special pains to have the dentist present in such operations where there is any question of his liability in the case.

Dr. Harmon Smith, New York City: I wish to call attention to a case more or less similar to the third case mentioned by Dr. Shurly. The patient was having his back upper molar tooth drilled by a burr, as represented in the third case. The dentist dropped the burr while the patient was in a semirecumbent position with his head backward and drawn to one side. The dentist took up a pair of forceps and when he went to find the burr it was gone. He became alarmed and told this gentleman to have an X-ray taken. The gentleman felt no inconvenience, and instead of that went to a board meeting downtown, and in due course of the day had the X-ray picture taken. It showed the burr in the upper left lobe, and he was then taken to the General Memorial Hospital. He was bronchoscoped, and the bronchoscopist attempted to remove it. He tried for a while and did not succeed, and asked me to come up. I failed, and we decided then to have Dr. Jackson see the patient. In the meantime another gentleman in town was suggested, and he came and failed. Dr. Jackson came on, and he had another X-ray taken to see exactly where the foreign body was located, and he put it down as a case in which it was impossible to obtain the foreign body. It had lodged in the upper outer lobe of the lung. It was recommended by the surgeons that part of the lung should be removed, but the gentleman succumbed to the operation. The burr, however, was obtained.

DR. ALBERT C. GETCHELL, Worcester: I will add two cases to Dr. Shurly's; one following extraction of a tooth under ether. The patient was operated upon and recovered, and the bill was paid by the society by whom the dentist was insured. The second case was a patient seen recently with a cough following immediately upon extraction of a tooth. This patient developed lung abscess, was operated upon and recovered, and then had a recurrence of the abscess.

DR. CARL E. MUNGER, Waterbury: I know of a case in which a dental burr was lost and the fluoroscopic view showed it just below the vocal cords. Tracheotomy was done and no dental burr was found. I should say that during the administration of the ether there was a good deal of struggling on the part of the patient, and that the burr was coughed up into the nasopharynx and lodged there. Next morning the patient found something in his mouth and spat it out. It was the dental burr. In this case the dentist paid the bill.

DR. BRYSON DELAVAN, New York City: I was familiar, as everyone else in our city was, with the case reported by Dr. Harmon Smith. The thing which impressed me most with regard to that case was the futility, as a general rule, of the radical operation for the removal of a foreign body. In looking over a long series of those cases it seems to me, while the observation may not be exactly germaine, unless the bronchoscope will succeed in removing the foreign body, the patient's chances of life are better if time is given before placing him in the hands of a surgeon, than if placed at once in the hands of a surgeon and an operation for the removal of the foreign body performed promptly. This was exactly my attitude at that time.

Dr. Emil Mayer, New York City: I recall the case of a gold crown of a wisdom tooth which was inhaled by a patient. With the valuable help of our member, Dr. Arrowsmith. I was able to remove this successfully. The case occurred about two years ago. One interesting point in regard to that case was that the anesthesia was rectal, which prevented anything like a struggle on the part of the patient, thus possibly dislocating the foreign body; and another point of interest was that as I removed the gold crowned tooth, Dr. Arrowsmith was standing guard over the patient, with a pair of forceps in his hand, and as that large crown was brought into the mouth and slipped from the grasp of the forceps, Dr. Arrowsmith promptly grabbed it and brought it out.

Dr. Robert C. Lynch, New Orleans: I would call attention to the case of a lady who was having a set of false teeth made and the dentist was using plaster of Paris as for modeling. He put in a cup filled with plaster of Paris and pushed this up, forcing her head back, and the plaster of Paris went down into her lung. This was five years before I made a bronchoscopic examination. I removed nine pieces of plaster of Paris from both lungs. I also found at that time a very marked constriction of the bronchials, and these I dilated, relieving the persistent spasmodic cough completely.

Dr. Stanton Friedberg, Chicago: We may divide the accidents of work on the teeth and other dental accidents into two classes: First, accidents that occur at the time of operation; and, second, accidents that occur from insufficiently attached or loosely attached crowns and bridges.

In one of my cases the cough came on immediately after anesthesia for extraction of a tooth. The cough persisted for a number of months, and the patient was sent to Colorado, where a picture was made, and she then returned to Chicago. There was a small abscess in the lower part of the right lung. The tooth was recovered by upper bronchoscopy, but the abscess persisted. The patient was under observation for three to four months afterwards, without perceptible improvement.

Another case was that of a man who in an intoxicated stupor loosened a bridge consisting of three teeth, which he aspirated into the left bronchus. A picture showed the bridge. On attempting to cocainize the larynx, hemorrhage of the lung started. The tube was introduced, but it was impossible to see anything. A second attempt was made under fluoroscopic bronchoscopy, and although the hemorrhage recurred the foreign body was extracted.

In the third case, that of an aspirated dental broach, upper bronchoscopy could not be done on account of the inability of the patient to open his mouth. In consultation with his surgeon, inasmuch as his symptoms were not acute, we decided not to do a tracheotomy, but to wait until the swelling about the jaw subsided. On account of the fine point of the broach the patient was advised to refrain as much as possible from coughing. Fortunately for him, however, the broach was coughed out several days later.

DR. JOHN F. BARNHILL, Indianapolis: Five years ago some one in Indiana consulted me concerning his daughter, who lived in New Jersey. She said she had had some sort of nasal operation, and on the way home was seized with asthma, and he wished to know what could be done. I suggested that possibly some foreign body had gotten into her larynx or trachea. Later he consulted me again, and requested that he consult Dr. Jackson. She started to Pittsburgh, and on the way coughed out a complete turbinated body that had in some way been inhaled during this operation.

DR. BURT R. SHURLY, Detroit (closing the discussion): I have nothing more to add except to emphasize the fact that there are really a great many of these cases, apparently. If we got them all together there would undoubtedly be a certain number of cases of lung abscess, the details of which have not been properly looked into, which would be proven to be of foreign body origin.

Sodium Bicarbonate in Ether Anesthesia.

BY GEORGE B. WOOD, M. D.,

PHILADEPHIA.

Modern methods of investigation have shown that both ether and chloroform anesthesia, when prolonged for over thirty minutes, are accompanied by a distinct lowering of the alkali reserve of the blood. It is probable that this mild transient acidosis is a most important factor in the production of postoperative vomiting. It has been further shown that the alkali reserve of the blood can be increased by the administration of sodium bicarbonate, and it is, therefore, a most rational procedure to use this drug routinely as a preoperative prophylactic measure against excessive postoperative vomiting.

DISCUSSION.

DR. EMIL MAYER, New York City: I would like to ask Dr. Wood which dosage of bicarbonate of soda he finds essential in children prior to operation—that is, if one must start with a certain amount of dosage, what practical dosage he has found beneficial.

DR. ROBERT C. LYNCH, New Orleans: I had a sad accident occur to a member of my family which brought this subject pretty closely home, and since that time I have used bicarbonate of soda as a preliminary to all ether anesthesia. Lately we have changed from bicarbonate of soda to citrate of soda as being a little bit more agreeable to the patients. We begin three or four days ahead of the time—that is, preceding the anesthesia—and give doses of bicarbonate of soda or citrate of soda, five grains, and of the former one-quarter of a teaspoonful three times a day. In those cases where we are suspicious of an inclination of the patient to develop evidences of acidosis, we follow the anesthesia always with a Murphy drip, five per cent, glucose solution, one-half pint. This gives our patients very much more comfort and freedom from vomiting. I think the thing is extremely important. It was suggested about three years ago to Dr. Peck, who adopted it. We find that during the intense heat we see many more cases of acidosis occurring then we do in the winter; and always during the spring and throughout the summer months, when we submit a case for general anesthesia, we never omit the preliminary soda treatment and the postoperative five per cent glucose.

We find, also, that the administration of codein and morphin after operation will increase the quantity of acetone in the urine, and predispose to the cyclic vomiting.

Dr. George B. Wood, Philadelphia (closing the discussion): In answer to Dr. Mayer's question, the dosage should be approximately one and one-half grains of sodium bicarbonate to each year of the child. It should be started the day before the operation, given preferably one-half hour before meals, the last dose being given one or two hours before the operation.

I have not been in the habit of using sodium bicarbonate after the operation unless there is evidence of acidosis, as manifested by constant vomiting or other symptoms, when it should be given by the "Murphy drip."

One other preoperative measure which I think should always be attended to is the proper feeding before the operation. The restriction of food should only be sufficient to assure an empty stomach when the ether is given. The most severe case of acidosis following anesthesia which I have seen was one in which the family physician had, without my knowledge, practically starved the child for twenty-four hours previous to the administration of the ether.

Removal of Foreign Bcdies From the Larynx, Disproving Previously Made Diagnoses.

By Hill Hastings, M. D.,

Los Angeles.

The rapidly growing use of direct laryngoscopy is showing up many incorrect diagnoses, especially in children, where foreign bodies were found in the larynx and trachea. The increasing number of them makes one feel that it is worth while to report all such cases.

Case 1.—Baby L., aged seventeen months, had been sick for a week with "croup," with gradually increasing obstruction to breathing. The father, a physician, and a brother practitioner, had been treating the patient with the feeling that the trouble was "croup," with slight bronchitis. No diphtheritic membrane had been seen in the throat, and cultures from the secretion had been negative for diphtheria. There had been slight respiratory obstruction and a little fever—100.6 degrees the highest. The baby was asleep in bed at the time of our consultation, and was breathing with audible roughness, but without cyanosis and without any considerable difficulty. The possibility of foreign body impaction was suggested, whereupon the father said that he dated the trouble to a little choking spell which the child had had when fed some soft boiled egg; but that the child had not really suffered much until two or three days later, when the increasing croupy cough and cry and a little fever had made him disregard the choking event. On waking the child its crying increased the dyspnea and brought on some cyanosis, which subsided quickly when the child again became quiet. Indirect laryngoscopy was a failure. The father preferred to await the result of X-ray examination and the use of simple therapeutic measures before allowing direct laryngoscopic examination. The X-ray examination was negative. The child's obstruction grew gradually worse, and direct laryngoscopic examination was agreed to. After a consideration of the danger of operating without a preliminary tracheotomy, it was finally decided to do a tracheotomy. With the use of Jackson's small-size laryngeal speculum, a piece of egg shell was found embedded in the larynx, between the cords, protruding into the glottis. The egg shell was easily remvoed. Convalescence was uneventful. The tracheotomy tube was not removed for three or four days because of the difficulty in breathing that resulted on attempts to do without it, which supported the contention that a preliminary tracheotomy was advisable.

The history of the second case was rather indefinite. Baby W., sixteen months old, was hurriedly brought from the country to the California Hospital because of great dyspnea. The child was already on the operating table when the writer first saw it. The only history obtained was that the illness dated back fifteen days to a choking spell that occurred while the child was sucking a piece of mutton chop bone.

Foreign body impaction was at once suspected. Immediate tracheotomy was done without any anesthetic. The patient was practically unconscious from the deep cyanosis. On opening the trachea, immediate relief was obtained and the acute pigeon-breast, tumor-like appearance at once disappeared. The end of a piece of bone was felt at the tracheal opening. The bone, rather firmly impacted above in the larynx, was removed by forceps. It was a large, rather thick sliver, about one inch long, sharp at its upper end. The child's fever and considerable purulent discharge and cough continued for a few days. Recovery was complete, and the patient was discharged on the sixteenth day.

DISCUSSION.

DR. JOHN F. BARNHILL, Indianapolis: I have seen two similar cases. One in a child that had been treated for about four days. This child had a safety pin just below the larynx, closed end down. Tracheotomy was done with easy removal of the pin. Complete recovery followed.

Another child, thirteen months old, with exactly such a history and in such a condition as the doctor described in his paper. The child was treated, as I remember, by four different physicians, all for croup. One believed the child had asthma. The X-ray showed a safety pin in such a location, which was easily removed by tracheotomy.

DR. HARMON SMITH, New York City: A case comes to mind of a patient six years of age, treated by six different doctors. It was treated for diphtheria and had antitoxins, had intubation done on two different occasions, and was finally sent to me by a prominent nose and throat specialist to remove a papilloma of the larynx. So I prepared, on his diagnosis, for a removal of a papilloma of the larynx. As I was going to remove it I saw the tip end of a safety pin lodged in the anterior commissure, wedged in between the two vocal cords After its removal there was no further trouble.

DR. THOMAS HALSTED, Syracuse: I had a case which was more or less similar to the egg shell case. This child was also treated for diphtheria and had antitoxin for two or three weeks, and then a diagnosis of a probable foreign body was made. That was before the days of bronchoscopy. Tracheotomy was done and three pieces of egg shell were removed; the child recovered.

Since the beginning of bronchoscopy I have operated through the bronchoscope two children, eight months of age, one in which we found a fish bone below the vocal cords, which was removed, and in the other a spicule of glass was found, but which was not removed. We got hold of it, but it slipped the forceps.

As long as fifteen years ago I reported seven cases of foreign bodies in children, all between two and three years of age, all seen and operated within a period of two and one-half years. Recovery of the foreign bodies in these seven cases took place by means of tracheotomy. Six of the children recovered, one dying of pneumonia.

DR. T. HUBBARD, Toledo: I have an X-ray plate which somewhat explains how foreign bodies can get into the larynx of young infants. This is a six-weeks-old baby, with a small, open safety pin. It was located in the posterior commissure, and was working down into the larynx.

There is another case I have in mind, of a fragment of hickory nut shell on the ventricle of the larynx, which by passing in pledgets of gauze and drawing up through the larynx finally released the hickory nut shell.

External Surgery of the Superior Maxilla in Treatment of Nasal Disease.

BY JOHN F. BARNHILL, M. D.,

INDIANAPOLIS.

The difference in the viewpoint and method of attacking nasal tumors of the nostril and its environment depends to some extent, no doubt, upon the degree of surgical training of each class of operator, and also upon the respective ability of each to accurately locate the origin of the growth. In case the origin is from the ethmoid, turbinates, septum or one of the walls of the nostril, the symptoms of early obstruction will cause the patient to consult the rhinologist, who, either from tradition or belief in its greater efficacy, will almost without exception attack from within. If, however, the growth begins without the nostril, the general surgeon often sees it, usually at a late state, and attacks it by those external methods universally approved by his branch of the profession.

Again, and rather frequently, malignant disease of the upper jaw attacks the alveolar process first, and from the fact that the earliest symptoms are neuralgia referred to the teeth, loosening of the teeth, and swelling of the adjacent alveolus, the patient is first seen by the dentist, who has been known to treat the teeth and even to extract healthy teeth, evidently in the belief that the disease is in some way connected with dental surgery.

A study of reported cases of sarcoma especially demonstrate on the one hand that rhinologists often have cauterized and snared at malignant growths in the nose whose undoubted seat must have been in some distant part of the maxilla, while on the other hand the general surgeon has been guilty of removing the entire upper jaw for a disease that had its origin in the nose or in the party line between the nose and antrum.

While it is probably true that the statistics of the rhinologist are better than the statistics of the general surgeon in the treatment of malignancy of the nose and its environment, these statistics are not entirely fair to external surgical methods. It is conceded by all that the length of time a malignant tumor has progressed prior to the operation has much to do with the end results of most operated cases. It is undoubtedly true that the rhinologist and the dentist see malignancy of the maxillæ at an average earlier time than does the general surgeon. Sarcoma or carcinoma develop symptoms of distress much earlier when originating in the nose or alveolus than when the origin is in the antrum. Antrum malignancy may progress many weeks or even months before there is external or nasal swelling and before pressure symptoms cause pain.

A candid view of the facts concerning the surgery of the upper jaw for malignancy is not altogether encouraging. The reasons for this are clear, and some of them have been stated. External surgery of the maxilla is essential, usually more essential, to cure than intranasal surgery. The rhinologist who pretends to do radical intranasal surgery should prepare himself to follow surgical disorders leading from the nose to any part of the upper jaw, if not, indeed, to wherever they may lead. When this is done, and when the diagnosis is made earlier than at present, great improvement of statistics may be expected.

All textbooks undoubtedly lay too much stress on total excision of the maxilla, for the reason, apparently, that it is presumed the disease has always advanced to a point that involves the whole upper jaw, or that any procedure short of total excision is inadequate.

One of the chief objections to the external surgery of the maxilla has been the deforming scars, the misplaced eye, and the palatal defects that result. Early and definite diagnosis as to the seat of the beginning of the growth, and an early studied plan of operation, will very largely avoid this objection. Heretofore the plan of operation has been too much a matter of doing one of two things in any given case—namely, some degree of intranasal surgery by the rhinologist, or complete removal of the jaw by the general surgeon. Earlier opportunity and sufficient thought bestowed on any given case on how best to break away from the traditional plan of total resection will in all probability find a satisfactory modified, yet thorough, operation to fit each condition present.

The writer has seen but four cases of undoubted malignancy whose origin was evidently within the nostril. Three of these cases were sarcoma, one epithelioma. All were operated intranasally, with one recovery—a spindle cell sarcoma of the inferior turbinated body. Two cases later extended to the ethmoid and antrum, were operated externally, one dying within about one year following the external operation, and the other within eighteen months. One case returned in the nasopharynx, and was so rapidly extending that the patient lived but a few weeks after the onset of the nasal ailment.

DISCUSSION.

Dr. Joseph H. Bryan, Washington: I am very much interested in this paper of Dr. Barnhill's, for I am in favor of external surgery in these cases. I had a case operated upon this spring, not a malignant growth, but an exaggerated fibromatous condition. The face was enormously distorted, the eye greatly displaced, and it was clearly impossible to remove it intranasally. I did the Moore operation, and was greatly pleased with the tremendous facility which it afforded for the complete removal of everything within the maxillary antrum, and also the facility with which it aids in removing not only the growth in the antrum, but the complete removal of the whole nasal wall, including the superior middle and inferior turbinate bodies. In this case, unfortunately, the under surface of the wound became infected and broke down. It healed thoroughly, and while the scar is somewhat on the lower border, it was more than a good result under the circumstances. I believe this operation of Moore is by far the best operation—that is, for complete eradication of all these growths which take place within this cavity. This was not a malignant growth, but the same thing would apply to any growth which takes place within that sinus.

Dr. Bryson Delavan, New York City: The reference to the treatment of nasopharyngeal fibroma leads me to say that I stand exactly where I did when I published the articles on that subject. Of course, there are exceptional cases where the growth has advanced far beyond what is usual; where it involves the sinuses to such an extraordinary degree that it is not practical to apply the treatment—the old fashioned at least thirty-year-old method of procedure of removal by electrolysis, which in the case of the less well developed growths has never been improved upon.

I have never heard of a case dying from the plan of treatment to which I referred, and I have not been able to find any in the literature where death has resulted, and the results are far better in every respect.

Hence, I want to again call your attention earnestly to the value of the electrolytic method of removal.

In any attempt to treat such cases with radium, it cannot be brought into communication with the growth. In those cases the best plan suggested has been the opening of the maxillary sinus, and the best avenue of approach seems to be through the roof of the mouth, where complete access can be gained to the maxillary sinus and where the opening can be kept patent. This is a great advantage, in that it enables the application of the radium to be made.

Dr. Hanau W. Loeb, St. Louis: I reported a case a number of years ago in which the tumor was removed by the electric cautery, and in which the hemorrhagic tendency was tremendously reduced by using the electrolysis method.

I may call attention to the ease of going into the nasopharynx by removing the posterior portion of the palate, as I did in a case of carcinoma in the epipharynx, a case not yet reported. The ease with which we were able to remove the mass from the nasopharynx was remarkable.

Dr. D. C. Greene, Jr., Boston: The subject as presented involves the field of rhinology occupied previously by general surgeons, and I would like to say a few words with this point in view.

As Dr. Barnhill has said, the tendency has been for general surgeons, in cases of malignant disease of the upper jaw, to perform a stereotyped operation, usually the classical operation, for removal of the upper jaw. In many cases such an operation removes a great deal of healthy tissue unnecessarily, and, what is worse, fails to reach the limits of the disease. It seems to me that we, as rhinologists, are especially qualified, by reason of our technic in intranasal examination, to carry out more careful observation and removal of the diseased tissue than the general surgeons.

I have been fortunate enough to operate successfully on three cases—and there has been no recurrence in either case—of fairly extensive sarcoma of the nose involving the antrum and ethmoid sinuses, in which a complete exposure of the growth was obtained by means of the Moure incision, so that complete removal of the tumor could be effected in each case. The results have been most satsifactory. One case operated on eight years ago has had no recurrence, another four years ago, and another one a year ago.

My point is that I believe it should be emphasized that we are better qualified to examine and treat these tumors surgically than the general surgeon, because of our general training and technic in intranasal work.

Dr. John F. Barnhill, Indianapolis (closing the discussion): If we can definitely demonstrate that the disease is in the floor of the nose or on the septum, it then seems, judging from statistics, that we are entirely justified in attacking the disease by means of electrocauterization. But if, as may happen, the disease has begun in the nasoantral wall, or has begun in the alveola and spread to the nose, or in the antrum and spread to the nose, or has begun in the ethmoid and spread to the nose, the rhinologist should recognize that fact, and then either send this patient at an early date to a surgeon, or, if he is himself qualified to deal surgically with it, he should do that. It seems to me that the time has come when we should not be guilty of attacking a great subject in a small way, as has been done in nasal surgery of sarcoma.

I emphasized, or tried to do so in my paper, the fact that the general surgeon has not gotten these cases in time to do anything else but make the kind of an operation that he has usually done. We have attacked this disease and worked at the job, as it were, until there was often nothing else to do but to pass it over to the other fellow, and the other fellow often passes it over to the undertaker. There was nothing else to do but to pass it on to the undertaker when it got so far along. But it is possible in nearly every instance, if we use some skill, judgment, care and patience, to make a diagnosis in these cases early enough and operate in time to cure at least many of these patients.

Report of Cases of Bilateral Abductor Paralysis of the Vocal Cords.

By Ralph Butler, M. D.,

Philadelphia.

The first case was eleven years old, and had congenital syphilitic cerebrospinal meningitis, causing ptosis of the right eyelid and internal strabismus. The biceps, triceps, and knee jerks were absent. The pupils were irresponsive to light and accommodation. The laryngeal symptoms, dyspnea and stridor began when he was six years old, but disappeared after three doses of salvarsan, to recur three years later. The second case was a man sixty-two years old with a neoplasm in the upper part of the chest and neck, including the thyroid gland, and causing almost complete occlusion of the trachea. He had been under observation for five months, and improved under mercurial inunctions. The third case was a fatal one following the removal of the thyroid gland.

The cases illustrate the greater danger from a sudden paralysis. The first two cases have been able to go about for many months with relatively little discomfort from obstructions which were little, if any, less than that which was fatal to the third, in which the paralysis was very rapid in its appearance.

Bert believes the greater mortality from sudden obstruction is due to reflex paralysis of the respiratory centers through irritation of the laryngeal nerves, and Krieger maintains that it is due to irritation of the cardiac branches of the vagus.

DISCUSSION.

Dr. Walter F. Chappell, New York City: I would like to call attention to the case of a patient, a man forty-five years old. His present illness began one month before admission, when he became hoarse. This continued and was followed by dyspnea, at first on exertion, later continuous. His only complaints were hoarseness and dyspnea. When he entered the hospital a tracheotomy was done at once, as the vocal cords were immobile and almost in contact. The Wassermann and X-ray of chest were both negative. Salvarsan was given twice and had no effect. Two weeks after admission, under suspension, the left vocal cord was removed with scalpel and punch. Three weeks later, as there was not space enough between the cords, the left recurrent laryngeal nerve was cut at the entrance into the larynx. In ten days the tracheotomy tube could be removed. When seen two months after the last operation, he was at work and breathing easily. Cutting the nerve not only allowed the vocal cord to recede into the cadaveric position, but caused an atrophy of that side of the larynx which gave still further space. The man was to report if he had any difficulty, and has not as yet, so it is presumed he is well. Several physical examinations and nerve reflexes were negative.

Dr. Greenfield Sluder, St. Louis: I would like to add the record of a case seen in consultation. A physician, twenty-eight years of age, who developed acute laryngitis, apparently a grippe, and with it a very violent dyspnea which, upon inspection, showed bilateral posticus paralysis. The cords were almost in the median line. The glottis was represented by a slit through which you could have dropped an old-time silver five cent piece. The recovery was not exactly uneventful. It required some six weeks, during which time he became excited about something and smothered almost to death. Nine or ten months later he developed another infection, laryngitis developed, and again posticus paralysis, and through the three attacks I saw him in consultation.

DR. BRYSON DELAVAN, New York City: Several years ago I was called in to see a case at St. Luke's Hospital suffering with dyspnea, and I found a marked abductor paralysis. I was obliged to do an immediate tracheotomy. The obstruction, however, was not entirely due to the laryngeal box. At a certain distance below the larynx, in the trachea, we also found constriction, and endeavored to overcome it by insertion of a long tracheotomy tube. The child died of exhaustion, and was found to have a large tubercular lymph node pressing upon the trachea, which was evidently the cause.

DR. W. B. CHAMBERLAIN, Cleveland: I have in mind such cases as occur in the early stage of locomotor ataxia. My attention was attracted to this a number of years ago by a patient who was found unconscious in the street and taken to the police station on the suspicion of being drunk. After the usual delay it was discovered that he was not drunk but ill. On examination we found complete abductor paralysis and other signs of locomotor ataxia. I then went over to the City Hospital and made a routine examination of all tabetics we had there, and was surprised at the number of cases in which we received no history of this condition, where we found paralysis.

Some few days ago I had a patient, a man about thirty-five years old, an Italian, who came with a history of dyspnea and difficult respiration, especially on exertion. I thought, of course, of the possibility of a foreign body, but examination showed bilateral abductor paralysis. I wished to refer the man to the hospital, but he disappeared. Some four weeks later the consultant told me that he found the man in an acute attack of dyspnea from which he succumbed in a short time.

Some Clinical Observations on the Lingual Tonsil.

BY GREENFIELD SLUDER, M. D.,

ST. LOUIS.

The diagnosis of lingual tonsillitis is simple in acute follicular cases. Should it not assume the follicular markings, it is often overlooked, particularly when the mass is not enlarged. It is recognized under these conditions by its color alone. The mass may be much enlarged in acute or chronic cases, which is, of course, easily recognizable. Not so easily interpreted is an occasional small slightly reddened follicle. These are often the origin of the symptoms.

The prognosis for singers and speakers, according to the writer's experience, should be guarded. A lingual tonsil which easily becomes a disturbing factor, either from the work of singing or speaking or from infection, must be considered most seriously. He does not believe that the singing or speaking voice can be developed to any great extent under these conditions.

The treatment of lingual tonsillitis in the acute follicular stage is like that for the faucial tonsils under like conditions. For the subacute or chronic state, with or without enlargement, nothing has been so satisfactory as applications of a small amount of silver nitrate saturated in fifty per cent glycerin. Salicylic acid saturated in ninety-five per cent alcohol is helpful, and does not taste so unpleasant. These may be made daily or as seldom as ten days. For the enlargement, galvanocautery destruction has seemed best. Hemorrhage following surgery of the lingual tonsil is more difficult to manage than any in the upper air passages.

Many of these observations have been made and recorded in more or less this form. The association of lingual tonsillitis with thyroid gland disturbance and with glossodynia, he believes has not hitherto been recorded.

It is his interpretation of clinical laryngology that the lingual tonsil plays a major rôle.

DISCUSSION.

DR. ALBERT G. GETCHELL, Worcester: I am very much interested and impressed with this account of the lingual tonsil. The disease undoubtedly has certain lesions that we probably do not appreciate. There is no question that there is definiteness about it and its lesion. I would like to call attention to three points which have impressed themselves upon my experience: first, its relation to the nervous system; second, to hemorrhage; and third, to cough.

There is no question but that trouble in this region would cause cough, but still I think that continued cough should not be attributed to such a lesion without a most thorough examination of the lungs. Simple examination of the sputum, without any examination of the lungs whatever, will often show the real cause of the trouble.

DR. HENRY L. SWAIN, New Haven: I have often seen how many times the sources of little lesions and the inconveniences that patients have suffered from could be attributed to and explained by conditions found in the lingual tonsil. The circulation of the lingual tonsil is peculiar—or, rather, its relation to the veins of the base of the neck is peculiar. The veins from the surface of the tongue gather together into certain large vessels deep down in front and underneath the lingual tonsil. The sudden occurrence of a feeling of fullness is perfectly explicable when you consider these venous branches which gather together underneath the lingual tonsil.

It is equally a fact that emotional stress, like an irritable plexus, is sometimes evidenced by this feeling of a lump in the throat, and this condition which I have described explains it.

It is perfectly possible to have a lingual tonsil cough and a bronchitis existing in the same person. If one treats the bronchial part and not the lingual tonsil, he is falling far short of his duty.

DR. J. PAYSON CLARK, Boston: My experienec has been that the enlargement of the lingual tonsil is observed most frequently in middle aged women. The enlargement of the lingual tonsil in children, in my experience, is a very rare condition.

I have had to remove the lingual tonsil in several cases, and have used Myles' lingual tonsillectome. I have also used the wire loop. If this loop can be used it is better, because you are less liable to have hemorrhage. I have not had hemorrhage in any of my cases. I remember being called late one night to see a case for bleeding, where another physician had removed the lingual tonsil that day. The bleeding was not violent, but persistent, and I had a little difficulty in stopping it.

DR. THOMAS HUBBARD, Toledo: I think there is no question about the intimate association between acute throat conditions and acute thyroiditis, because I think we find many cases in which we are able to successfully treat the subacute chronic throat because it is most helpful in the reduction or checking of acute thyroiditis.

In a ten-year-old child, four years after removal of her tonsils and adenoids, her lingual tonsils were found enormously enlarged. There was no suggestion of recurrence of either tonsillar or adenoid tissue, but these large masses were a mechanical obstruction to swallowing, and also made the swallowing more or less painful. To prove that, I clipped off portions of the lingual masses, and following that there was no inflammatory reaction.

Dr. Robert C. Myles, New York City: There are several points about the lymphatic tissue at the base of the tongue. It seems to me that a very interesting one is whether the removal of the tonsils in early life does not by some vicarious attempt on the part of the lingual tonsil to supply the physiologic function of the tonsil, result in their becoming hypertrophied in this attempt.

It has been my habit for many years to remove lingual tonsils in a little different way from that originally indicated. On account of some hemorrhages and cicatricial tissue, I have tried to devise some method of obviating both; one was to take out every other lymphatic tonsil with a guillotine and leave an intermittent one. That aided me very much in relieving the scar tissue. I have also left islands of tissue. In using the guillotine one should not press too deeply. If you go beneath the tonsil you are more liable to have hemorrhage than if you put the proper pressure on the guillotine, which makes it possible to remove as much as you may elect.

Dr. Burt R. Shurly, Detroit: The importance of this relationship of Waldeyer's ring to the thyroid is certainly a very exact and definite thing, but the key to the whole situation, to my mind, is infection. And while this paper which was referred to by Dr. Sluder was written five years ago, I have followed up carefully a large series of cases since that time. Of course, as Dr. Sluder says, no one expects to treat cases in this manner and not have the proper surgery of the thyroid attended to when that is necessary.

In regard to cases of hemorrhage from the throat, I think perhaps there are a great many mistakes made by not properly examining the chest. We know full well that twenty-five per cent of all cases of pulmonary tuberculosis have hemorrhage at some time or other during the course of that disease, and it seems to me that it is just a matter of not thinking of that. We should always have a very thorough and complete chest examination wherever there has been hemorrhage from the throat.

DR. JOHN MACKENTY, New York: I believe that any infection in the throat may have secondary expression in the thyroid. This was brought home to me very forcibly by the case of a man with a foreign body in the esophagus. I put in the bronchoscope and failed to get the foreign body, but found the spot where the body had rested. This man was very ill, with a high temperature and pulse, and had a tender enlarged thyroid gland. I was positive there was pus in the neck, and opened the neck and went down and found no pus. I then opened the thyroid gland, and in the center of the gland I found a large abscess which was secondary to the condition in the esophagus.

On another occasion, following the removal of the tonsil, there was considerable local infection and the patient developed acute thyroiditis. So I believe we may say that any infection in the throat or nose may have secondary manifestations in the thyroid, either simple or suppurative.

With regard to the question of bleeding of lingual tonsil, I have found benefit from forcibly pulling the tongue forward and holding it in that position for a time.

DR. MAX A. GOLDSTEIN, St. Louis: In reference to Waldeyer's ring, I think it has been rather insignificantly looked into, and Sluder's observation about the relationship to systemic infections taking place through this small lower lymphoid mass ought to give us much food for thought. We are doing a lot of operative work on the faucial tonsil, and our enucleations are coming thick and fast. We know that even when the faucial tonsil has been thoroughly encapsulated, we do have recurrences of systemic infections; and it is likely that many of those infections come, not through the lingual tonsil, but through the lymphoid masses which lie about the pillars. There is some possibility that such observations as were presented by the essayist may give us an opportunity to think of this radical surgery when the time comes for the pendulum to swing the other way.

Dr. Swain has pointed out on previous occasions that the mass of lymphoid tissue is rather dependent for its pathology and inflammation more on the intense venous relationship than on the lymphatic. I think that varicosities are sometimes as frequent in lingual tonsil as are the acute and chronic follicular inflammatory reactions, but those are things this paper has brought forth, and I believe that the development of this particular phase of Waldeyer's ring will give us a little closer insight into the whole pathology of this lymphoid area.

DR. GREENFIELD SLUDER, St. Louis (closing the discussion): I feel that a very substantial addition to the paper has been given by Dr. Swain in his explanation of the globus hystericus. I did not know the network arrangement of the veins beneath the lingual tonsil, and it is not recorded in any of the anatomies I know of.

Dr. Clark spoke of the enlargement of the lingual tonsil in children being rare. That is not my observation. The enlargement of the lingual tonsil in children is a very frequent manifestation.

I have seen the faucial tonsil take on the recurrent inflammatory reaction with sore throat after faucial enucleation.

That the chest must be investigated in cases of cough seems to me to be self-evident.

Some Points in the Surgical Treatment of Goiter.

BY J. E. MACKENTY, M. D.,

NEW YORK.

The writer emphasizes the need of laryngologists, and especially the younger men, to take up the surgery of the neck.

He considers all treatment with rest as a basis as deceptive, and the delay engendered pernicious. He does not, however, advocate surgery in all cases. In all progressive cases, short of already advanced toxicosis, some surgical effort should be undertaken, such as boiling water injections into the gland, ligation of one or more poles, removal of half the gland, with or without ligation of the remaining poles, etc.

In well developed and progressive toxic goiter nothing short of the ablation of three-fourths to seven-eighths of the gland can be relied upon to effect a cure. No operation should be considered until after the patient has been under observation in bed for a length of time, and has undergone a careful general examination.

Clinic calorimetry is now used to differentiate the active periods of the disease and to accurately determine the degree of benefit from any surgical or medical treatment. Early operation is most important. Local anesthesia should be rarely used, and never for any extensive operation. Deep anesthesia is not requisite. Rapidity of technic must be combined with gentleness of manipulation or it is valueless.

By complete operation is meant removal of from one-half to seven-eighths of the gland. In operating the four important things to be considered are: The parathyroids, recurrent laryngeal nerves, hemorrhage, and the amount of gland tissue to be removed.

These are considered in detail. Free drainage with a liberal opening is important, and permits continuous saline irrigation through the tubes, which is often exceedingly valuable.

Thyroidemia may be avoided by attention to the following points: Careful preliminary treatment, rest, overfeeding, etc.; the reduction of a minimum of fear on the part of the patient, light anesthesia, nontraumatic surgery, the judicious selection of the proper operation and correct time of operation; alkalies and water before operation, etc.

The following types of hyperthyroidism are bad risks:

1. Where the disease is progressive in spite of rest.
2. Cases showing no remissions.
3. Appearance of psychosis.
4. Very active symptoms in cases with small glands.
5. Marked exophthalmus.

No case should, however, be refused some form of surgical help, providing it can be established that the disease is not in its terminal stage.

DISCUSSION.

DR. JOHN F. BARNHILL, Indianapolis: It would be a fatal mistake to attempt any sort of operative procedure in a case in which the heart is so dilated or hypertrophied as not to be able to withstand the operation. The mortality from simple cases ought to be pretty nearly one hundred per cent.

I know of no other operation in surgery, except brain surgery, where a competent anesthetist is more useful than in this operation.

Every precaution must be taken to preserve the parathyroids, and this is best done by leaving a portion of the capsule posteriorly. The capsule in this operation I believe is the anatomic structure which must be borne in mind more than anything else, and if one loses his capsule and works outside the glandular capsule, he is lost for the whole operation, and he cannot, therefore, be certain as to whether he will injure the nerve or remove one or more parathyroids. Hence, his procedure must keep the surgical and glandular capsule in mind all the time, and if he works between them it is almost impossible either to injure the recurrent laryngeal nerve or to take away the parathyroid glands.

The patient is already, in toxic cases, badly run down, and, therefore, clean and bloodless surgery here is the thing. Shock does not occur often if you do not lose much blood.

The amount of a gland to be removed is an important question, requires the greatest knowledge, and it is here, I think, that consultation is often necessary. To remove three-quarters or seven-eighths of a gland that does not need that much taken away is a mistake. To leave it if seriously diseased is equally a mistake.

There are often other glands involved along with this gland, and if that fact is not ascertained before operation and operation undertaken, the operator will almost certainly have a death, because to remove a toxic thyroid does not in such instances cure the case.

The gland must not be handled roughly or pulled up unnecessarily with heavy instruments by the operator or those helping him. It must be handled gently, because if there is toxemia there may because of rude handling of the gland be unnecessary toxicity after operation.

DR. THOMAS HUBBARD, Toledo: I see many of these cases before and after operations with reference to voice interference and difficulty in respiration. Fortunately, most of the cases in which there is an impairment of the vocal cords, usually unilateral, improve within a reasonable time after operation. In others a supplemental action takes place in the opposite vocal cord, ultimately producing impaired voice, with permanent impairment of movement of the cord on one side.

DR. HARMON SMITH, New York City: I have tried to ascertain in how many cases the nerve was involved previous to or during the course of the operative procedure. I have reached no decision which enables me to make any statement relative to the positive number, but a great many were involved previous to the operation. There was a paresis and not a paralysis. I expect to make some reports relative to it in due course of time.

DR. JOHN E. MACKENTY, New York (closing the discussion): The reason for two tubes is that when you take out more than half the gland you have the trachea dividing the field. If you wish to secure complete drainage, you must put a tube on either side of this tube.

I believe the involvement of the laryngeal nerve interferes to some extent with the finer quality of the voice—as for singing or public speaking.

I have injured the nerve on one side and had impairment of the voice for a fair length of time, which was always recovered from, so far as speaking was concerned.

The Mayos report considerable percentage of nerve involvement before operation. I have examined cases pretty carefully, and have not been able to corroborate this. In a few cases there seems to be some lack of proper tone or movement of the cord, but not to the extent the Mayos report in their examination of cases before operation.

Discussion on Report of Council of National Defense.

DR. CHARLES W. RICHARDSON, Washington: We, as citizens of the United States, have benefited to an unusual extent from our citizenship in this country, more so than citizenship in any other country as individuals. We have profited by this; our success has been great, our comforts have been manifold, and it is no more than right that we should assume the duties and obligations of citizenship, and offer our services fully to our country, and encourage all those with whom we come in contact with this spirit of enthusiasm. We must stand, gentlemen, for our country, for democracy, and for the suspension of this horrible war, as soon as we can; and by giving aid and comfort and doing our utmost by enlisting ourselves and causing others to enlist, we can bring this about.

DR. HARRIS F. MOSHER, Boston: What the government asks of us today is a wholesale coming into their ranks without any reservation whatever. They say they can make no promises. On the side they intimate that perhaps, probably even, the special man will be used for special work, but no promises are given, so that you have to make up your minds whether you will go on with the feeling of a slacker and wait until you are asked to do special work, or go in today and run your chances of being put to work with which you are unfamiliar. The only way out of that is to stay in this uncomfortable position until some action is taken toward the forming of a special base hospital. When you read the report, if you do, you will find that was recommended. If you talk with the men from Washington you will find they feel that will come. Major Lister said that will probably come, if Pershing carries over the amount of men he is supposed to carry over, within the next six months. Until that time I am unhappy, and I feel very many of you are unhappy. I do not know what to do. It is a much easier thing to go in and a much harder thing to stand out. I for one am going to stand out until the time comes to go in and do the work I think I am more able to do.

DR. ROBERT C. MYLES, New York City: I have had some very peculiar experiences lately at the New York Polyclinic where we have three hundred specialists, and a lot of them have joined this corps, and I wish you could see some of the work they are doing. It is so far removed from their specialty that they know nothing about it. It is simply ridiculous. I do not know what I could do if I was sent down to the Mexican border to treat dysentery. According to what we hear, that will all be corrected later. There ought to be some action or activity, some committee formed or appointed with a medical officer to plan some law to relieve the situation. The eye and ear are two of the most important organs in war.

DR. JOSEPH H. BRYAN, Washington: This is a very important matter for us as citizens and as specialists, but we must remember that we are at a very grave crisis, and every man, young and old, every woman and every child, has got to come forward and do something. This is no child's play. This war, according to the best thinkers and the best observers, is not going to be over this fall, this winter, or even next, and every resource of this country has got to be utilized. I have gone into the Officers' Reserve Corps, having served some years ago in another branch of the service. Of course, it is a very hard thing to do, to go in and abandon the care and responsibility I have, but nevertheless I feel that having served once it is my duty to go there again. I have gone in voluntarily and am willing to accept any position. I have accepted a subordinate rank; I can serve as Captain just as well as Major, and am perfectly willing to do that, but we are all in bounden duty to do something to help out in this crisis. On the other hand, I think if the matter is brought very forcibly to the attention of the Surgeon General of the United States Army by these various societies, representing that men of experience, men who have developed these specialties of the eye, ear, nose and throat, are best fitted to work in the base hospitals or some hospital devoted to the treatment of diseases resulting from injuries, and that they can best serve their government if their services are utilized along these lines. I am sure the Surgeon General will treat you courteously and possibly do something along those lines. Of course, you must remember that the war department has been working on a basis of a hundred thousand men, and has to work now on a basis of over two million men, and have not got the system or force to combat these difficulties. They are swamped with work at present. But if we will take the stand and assert that our services are best utilized along the lines on which we have been working, I believe some impression will be made upon them. In the meantime we can all go in and serve our country, to the best of our ability.

DR. BURT R. SHURLY, Detroit: It will undoubtedly be necessary to establish a number of base hospitals of the eye, ear, nose and throat. We have one by way of example in the British army now located at Folkestone, which has done very wonderful service for the British army. If we could have a base hospital on the foreign service, and one in this country, or more as will be necessary, and organize the eye, ear, nose and throat hospitals of the various large cities as they are already organized, and have a definite method of referring all cases from these five hundred bed base hospitals of the general base hospitals to the special hospitals, we would undoubtedly be able to organize and establish a splendid service for the army of the United States at home and abroad.

It seems to me that this wonderful scientific usefulness simply requires organization and authority. Without the authority of the Surgeon General's office we can do nothing whatever, because there must be a definite plan of work, and the organization must be a definite one, and follow along a very definite plan, which has been worked out with very great detail by the Surgeon General's office.

Undoubtedly, the organization and equipment of an eye, ear, nose and throat hospital would require special organization and equipment. These are things which have never been done before in this country. Therefore, it does seem to me the time has come when a committee from these various societies should organize and send out at least two base hospitals. Therefore, there are forty-eight highly trained efficient specialists to care for certain cases from the various base hospitals which the ordinary eye, ear, nose and throat man on the staff of this hospital is not able to properly care for.

Dr. Joseph H. Bryan, Washington: I would like to emphasize one fact. In case this committee is appointed, and I hope it will be, the matter can be brought directly to the attention of the Surgeon General, and the fact emphasized that the men who are highly trained in the various specialties will be wasted if they are sent on the ordinary staffs, and the point made that now there is actually a very great waste of good material to be sent on duty of that kind when the younger men can be utilized for that purpose, and that we as highly trained specialists should be called upon to do the work as outlined by Dr. Shurly.

Dr. Charles F. Richardson, Washington: If we appoint this committee, which I hope will be done, I am sure the Surgeon General and assistants, both of the army and navy, will listen to that committee and will do all that that committee wishes—that is, within reason.

Every man knows what his duty is to himself, to his family, and to his country, but there is one fact that stands paramount. The example of the class of men that represents this society to the younger medical men throughout the country is a great one, and should not be lost sight of. If you can go about among the younger medical men and say, "I am already in the service," that young man is going to think, and he is more apt to offer himself to the service of his country if he knows that we have already offered ourselves freely to our government, to be used as the government sees fit. And I hope that the men of this society will offer themselves freely for the service of their country to be used as the army authorities may see best and fit to make use of them.

DR. D. BRYSON DELAVAN, New York City: I represent that type which does not know just what to do. I do not know what my place is. For a year and a half I struggled to establish a hospital in Paris for the repair of injuries of the head and face. At the end of the first year sixty thousand men were needing repair in that department alone, simply from France and Belgium. I am chairman of the Executive Committee of the American Red Cross Hospital in Paris, managed by Dr. Blake. The nearest member of my family last week got an ambulance unit that is on its way to the front, and each week a certain number of us go to the Naval Recruiting Station and examine ears and throats. In other words, not knowing what to do, like all the rest, we are willing to take the first thing that comes to hand and do the best we can. Dr. Shurly has touched the keynote when he said that organization is needed.

DR. MAX A. GOLDSTEIN, St. Louis: Without much circumlocution I would suggest that there be a propaganda of education to put us all in a position to study the question as carefully as we know how. This might be developed by the committee already urged. Personally, at the present time, I should like to place at your service for this organization and its committee, the *Laryngoscope* and its editor, for any publicity work you require whenever and as often as you require it, and to place at your disposal furthermore the mailing list of the office of the *Laryngoscope,* which includes the names of three thousand men of this country.

Final Conclusions Regarding Amputation of the Epiglottis for Tuberculosis.

BY LORENZO B. LOCKARD, M. D.,

DENVER.

Amputation of the epiglottis is as safe a procedure as tonsillectomy. In over four hundred cases, in the majority of whom vitality was reduced to the lowest ebb, not a single direct fatality resulted.

The objection most frequently advanced is that no operative procedures are justified unless the lesion is so circumscribed as to be capable of complete excision.

It must be borne in mind that in nine cases out of ten the sole object is palliation, and usually in patients upon whom all other methods of treatment have failed.

Even when a cure is considered possible, removal of all involved tissue is not invariably essential to success. It has been demonstrated repeatedly that when an epiglottis is universally infiltrated, and only the upper half or two-thirds is removed; the stump rapidly recedes to normal. It is rare that the disease recurs in the stump.

Healing of the stump is usually rapid and complete, regardless of the extent or rapidity of progress in the complicating pulmonary and laryngeal lesions, hence the question of advisability of operating in this manner hinges upon two questions: in incurable cases what amount of relief may be anticipated, and in cases otherwise hopeful, what influence will the operation have upon the accompanying laryngeal lesions?

A number of patients are living upon whom the operation was performed from ten to twelve years ago, as a palliative procedure, and in whom the resultant unexpected improvement in lungs and larynx was so complete that eventual arrest ensued.

The improvement in accompanying lesions can be ascribed in large part to the same influence that occasions pulmonary betterment: the removal of pain, increase in nourishment taken, improved sleep, and lessened cough.

Another important factor is the increased accessibility of the larynx to treatment. After the epiglottis is removed it is often easy to destroy by galvanocauterization lesions that were previously completely hidden. It is a fact that a surprising subsidence in these accompanying processes is frequently observed.

The chief indication for amputation, however, is and must remain, the relief of pain, without thought to the eventual cure of either laryngeal or pulmonary diseases.

The one great contraindication, in the author's experience, is that form of epiglottic involvement, either infiltrative or ulcerative, in which the process is beginning to involve the base of the tongue or the pharyngoepiglottic folds.

The entire lateral walls and base of the tongue may and usually do break down within a few weeks after the very first signs of disease become manifest. In these cases only is amputation absolutely contraindicated. In all others, if pain exists and is uncontrollable by other treatment, excision is advisable. No bad effect upon the general health has been observed. Complete anesthesia can be obtained, and the operation itself need not, in the average case, require more than a half minute.

Ozena and Asphyxiating Gas.

By MARCEL NATIER, M. D.,

PARIS, FRANCE.

The writer finds a melancholy pleasure in presenting the lamentable history of a young soldier, in that he feels that the recital of his case goes to prove conclusively that ozena is but a local manifestation of a constitutional condition, a fact which Natier had repeatedly maintained in previous communications.

In October, 1915, a soldier, twenty-four years of age, who had always been in perfect health, no hereditary or venereal affection, received five bullet wounds and was rendered unconscious by a bomb of asphyxiating gas. His mask had dried up and was valueless. He recovered consciousness only to fall into repeated syncope. He was carried to the rear, transferred to Vitry le Francois, where he had to remain three weeks because of his febrile condition. He suffered most excruciating pains from his nose to the bifurcation of his bronchi, and received special care. Five weeks after the accident he was removed to Paris, to a base hospital, where he remained six months. During all this time he could not swallow except with most excruciating pains. He then was admitted, March 18, 1916, to the Salpetriere, where his extreme muscular weakness, respiratory troubles, ozena and vomiting were noted. On October 25th the writer first saw him, and noted remarkable collapse of both alæ, ozena active, marked anemia. The collapse of the alæ caused insomnia and mouth breathing, with pain. To relieve this the patient put pieces of a match in each nostril on going to sleep. The ozena was noticed three or four months after the accident for the first time. Irrigations with warm salt water, while causing pain, was followed by amelioration.

As the patient was always well until his injuries, the writer feels justified in tracing the causal factor of his ozena to the asphyxiating gas and the subsequent functional disorders.

Ordinarily ozena is considered inseparable from old and far advanced atrophic rhinitis traceable back to the first years of life, the atrophy slowly developing.

In this soldier the determining cause was at the time sudden and violent, the effect cruel and deplorable. That the unfortunate man did not succumb immediately or in the general delay at the arrival of succor is marvelous. The poor fellow was fortunate in that he did not develop tuberculosis, as thousands of others do. The patient recovered.

The writer says, in his opinion ozena is not a true mobid entity, idiopathic or real, but a unique and always a symptomatic expression of a profound localized disturbance of the general health. Once again it is verified, and supports the assertion that ozena is but a consequence, distant more often, but also under color of exceptional events of the physiologic calamity. The cause of the latter is of less importance. Its intensity merits consideration.

This truth admitted and its verity visible, impresses him. We are compelled to discard, as he has always done in his studies of the subject, the various theories more or less fantastic, and in particular the microbic theory, invoked to explain the production of ozena.

(1) Sarcoma of the Nasal Septum; (2) Laryngoepiglottidean Cyst.

By Max A. Goldstein, M. D.,

St. Louis.

Male, aged fifty-six years. Eighteen months ago began to be troubled with occasional obstruction of left side of nose, continuing about a month, followed by relief and then recurrence. No history of epistaxis. Latterly two or three nosebleeds, not very profuse. No headache or pain of any kind. Loss of fifteen pounds in weight in six months.

Nasal Examination.—Mass on septum obstructing left side. Right surface of septum infiltrated and thickened, causing some obstruction on that side. Nasopharynx negative. Throat negative. No evidence of involvement of the accessory sinuses.

Operation.—September 26, 1916, mass, together with the entire septum, being extirpated. Recurrence after one month. Histologic examination of mass showed round cell sarcoma.

A Case of Laryngoepiglottidean Cyst.

Boy, twelve years old, came under observation September 27, 1915. Had been hoarse since he was one month old. This hoarseness had been ascribed by family physician to a "cold." No laryngologic examination had at any time been made. The condition seemed to remain quiescent all these years until shortly before the boy was brought to the writer for examination, at which time he had become very dyspneic. The dyspnea was quite marked; patient anemic and frail, not cyanotic, and unable to speak above a whisper. There had not been much difficulty in swallowing. No regurgitation of food. Laryngeal examination showed a mass the size of a walnut on the left side, involving the laryngoepiglottidean region. There was no fever. Mass was yielding to touch and could easily be palpated with the finger. Mass incised, the contents consisting of clear, yellow, sticky fluid.

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